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RESEARCH REPORT

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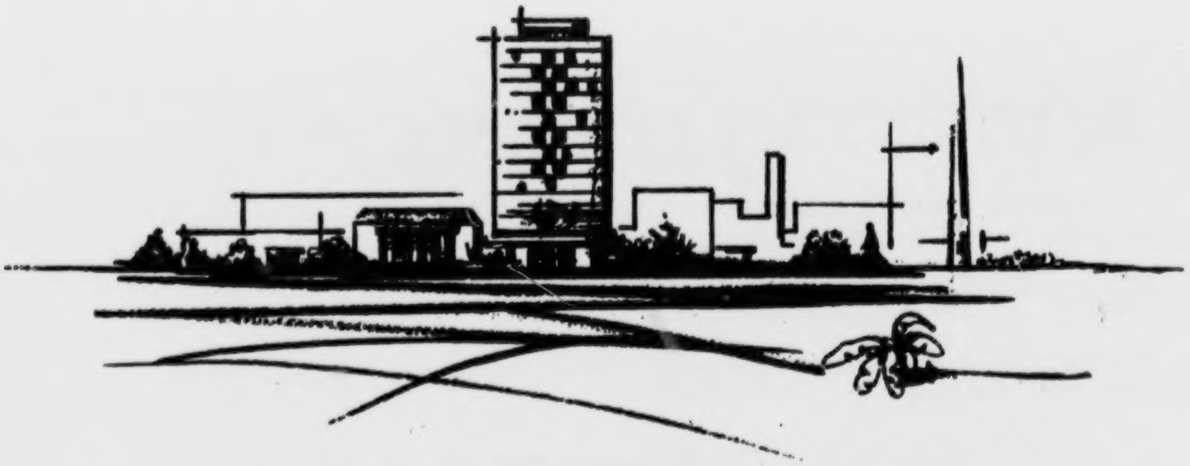

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The lead model case study was undertaken to serve as a working example and to evaluate the technical, intelligence, and project information system under consideration for EHS. Occurrence and use patterns for lead within the U.S. were examined. The major categories of uses in decreasing order were production of storage batteries and accessories, gasoline antiknock additives, pigments, ammunition, solder, cable covering, and caulking lead. Leaded fuels are the major source of environmental lead. Pediatric plumbism, the most significant acute health problem, arises from lead used as a pigment. The economic aspects of this problem area were investigated. While lead is very slowly absorbed from environmental sources, its gradual accumulation in the body is the basis for progressive toxicity. Additional research is required to determine the chronic effects of lead in the environment on health, and better diagnostic and therapeutic techniques are needed in support of this research. A mathematical model was developed to represent the environmental transport of the stressor lead from several sources with the subsequent intake of lead by man. This model can be used to assess the impact of contemplated actions on lead body burdens, to define R&D requirements, and to identify the needs and locations for monitoring and surveillance. A categorized bibliography for lead is included in this report.

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FINAL REPORT

on

TECHNICAL, INTELLIGENCE, AND PROJECT INFORMATION SYSTEM FOR THE ENVIRONMENTAL HEALTH SERVICE
VOLUME III. LEAD MODEL CASE STUDY

to

U. S. DEPARTMENT OF HEALTH,
EDUCATION, AND WELFARE
ENVIRONMENTAL HEALTH SERVICE

June 29, 1970

Contract No. CPS-69-005

by

Garson A. Lutz, Arthur A. Levin, Sanford G. Bloom,
Kaj L. Nielsen, Jack L. Cross, and David L. Morrison

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FOREWORD

This report is one of five volumes that were prepared for the Environmental Health Service under Contract Number CPS-69-005. The objectives of this project were to examine research and development program planning needs within the EHS and to develop a management assistance system for the EHS. Since a well-organized and readily accessible information base is essential to planning, existing information capabilities and facilities within EHS were surveyed and an environmental health information network was devised. Two model case studies for the environmental stressors, lead and persistent pesticides, were performed to serve as examples during the project and to exercise the element of the management assistance system.

The reports in this series are:

Volume I.	EHS Management Assistance and Planning
Volume II.	EHS Information Network Analysis
Volume III.	Lead Model Case Study
Volume IV.	Pesticides Model Case Study
Volume V.	Directory of EHS Information Facilities With Selected Supplementary Resources

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MANAGEMENT SUMMARY

The lead model case study was undertaken as a part of a project by Battelle Memorial Institute to investigate and develop an operational plan and staffing table for a research and development and program management and planning assistance system for the Environmental Health Service. The effectiveness of the system was tested by the case study, and areas of improvement were detected. The lead model case study was to provide examples for the types of information on environmental stressors that are currently available and to explore in a preliminary manner the R&D efforts needed to provide additional data to eliminate or control the hazards from lead in the environment. By selecting a specific stressor for study, mathematical modeling, priority rating, and the environmental stressor concepts which were evolved as a part of the overall management assistance system could be demonstrated and evaluated. The lead model case study also represented a comprehensive review of the environmental aspects of lead.

Lead usually occurs as either sulfide ores (galena, PbS), or oxide or carbonate ores (anglesite, $PbSO_4$, and cerussite, $PbCO_3$). Minerals of iron, zinc, silver, copper, gold, cadmium, antimony, arsenic, bismuth, and others may be associated, in varying proportions, in lead ore deposits. The average grade of lead ores mined contains between 3.0 and 8.0 percent lead. Mining, smelting and refining, secondary recovery, and imports are the four main sources of lead in this country. In 1969, mine production of recoverable lead was approximately 500,000 (short) tons, with Missouri accounting for 350,000 tons and Idaho 65,000 tons. Mine production in the U. S. during the past 10 years has exceeded 250,000 tons annually. The 1969 U. S. supply from mining, primary refining, secondary smelters, and recovery from copper-based scrap amounted to about 1,540,000 tons. During the last decade, the U. S. supply annually has been near or beyond 1,000,000 tons. Extending this rationale to the domestic lead consumption of primary, secondary, imports, and lead in lead ore entering directly into the manufacture of lead pigment and salts, a figure of 1,333,000 tons is obtained for 1969. These figures suggest that, even though only a small percentage of lead may enter the environment from the production and consumption cycle, a certain background level of lead would be expected.

An examination of the use pattern for 1969 and the years immediately preceding indicates how lead is added to the environment. Although there have been fluctations in quantities in the use categories reported, there have not been any major changes in the categories themselves reported during the last decade. That is, the major uses of lead during 1969 continued to be (in decreasing order) in the production of storage batteries and accessories, gasoline antiknock additives (mostly tetraethyllead), red lead and litharge pigments, ammunition, solder, cable covering, and caulking lead. An inspection of the materials requirements and manufacturing processes for the lead-acid storage batteries, e.g., preparation of pasted plates, preparation and assembly of grids, and particle-size distribution and grinding of lead oxides, revealed cases of lead intoxication in 66 battery-based industries in Pennsylvania. The use of old battery cases for fuel and the resulting lead-containing ash also were identified as environmental hazards of air and soil.

Examination of the use pattern of leaded gasoline additives – tetraethyllead (TEL) and lesser amounts of tetramethyllead (TML) – indicated that leaded fuels continue to be a major source of environmental lead. Each gallon of today's gasoline contains about 4 grams of lead, which adds up to 700 million pounds of lead consumed annually according to gasoline sales. About half of the exhausted lead becomes airborne, that is, each year about 350,000,000 pounds of lead swirls into the atmosphere. The remainder is either not dislodged from the exhaust system or settles on the road surface or adjoining areas. In recent studies, leaded gasoline has been identified as the overwhelming contributor of lead pollutants to the atmosphere. Analysis of atmospherically precipitated samples of lead and other metals, collected by a nationwide network of 32 stations throughout the U. S., indicated that the concentration of lead in precipitation correlated with the amount of gasoline consumed in the area in which the samples were collected. The above two investigations suggest that leaded gasolines contribute notably to the environmental (soil and water) burden. Red lead and litharge-based paints were identified as an environmental hazard during shipscrapping because of the high temperatures involved in burning off (volatilizing) the spent paint.

Further exploration of the lead-pigment use pattern brought to light the ban on the use of lead pigments in indoor paints. More important, however, it revealed the many instances of pediatric plumbism in substandard housing areas of some of our larger cities, e.g., New York, Baltimore, Cleveland, and Chicago. Factors such as the elimination of lead paint from walls, continuous education and enforcement activities of the city health department, efforts of local pediatric services, establishment of comprehensive health care clinics for children in the susceptible age living in high risk areas, family planning, and an improved social climate, have resulted in a drop of about 50 percent (286 to 141) in pediatric plumbism in Baltimore during two consecutive 3-year periods (1964 to 1966 and 1966 to 1969). Chisolm points out that these corrective measures deserve attention when it is realized that total treatment costs for an institutionalized child suffering from permanent brain damage may amount to approximately $250,000 during a 60-year period.

An examination of the methods of obtaining lead and the diverse uses of lead suggested that the effects of lead on man may be exercised through contact, inhalation, and ingestion. In order to determine the stressor ⟶ source ⟶ transport ⟶ path ⟶ effect (on man) relationship, an environmental stressor matrix was formulated for lead, including lead alloys. The stressor matrix approach appears sufficiently flexible to collect and display information defining the effects that any stressor may exercise, since it permits specification of the medium, the compartment, the portal of entry, target site of action, consequences, and control.

Lead is very slowly absorbed from environmental sources, e.g., soil, water, food, and air, but the gradual accumulation of the element in the body is the basis for progressive toxicity. It was formerly believed that considerable amounts of lead could gain access to the body percutaneously, but this has been refuted; instead, inhalation and ingestion are the two main routes of entry. The average man ingests about 5 mg/yr from drinking water, 100 mg/yr from his diet, and about 15 mg/yr from respiration. Generally, industrial health authorities agree that lead concentration in respired air should be below 150 $\mu g/m^3$ and tetraethyllead (TEL) should be below 50 $\mu g/m^3$. Concentration of lead in ambient air may vary from approximately 0.01 $\mu g/m^3$ to 50 $\mu g/m^3$ (the latter along heavily traveled highways).

Although the principal site of lead in the body is the skeleton, many other soft tissues, e.g., brain, lung, heart, liver, kidney, spleen, and muscle have been found to contain 20 to 120 µg of lead/100 g fresh tissue. Teeth are confirmed to have a higher concentration of lead than any of the bones, and the average lead concentration shows an increase with age, e.g., <10/years, ca 17 µg/g of ash, and 50 to 60 years, ca 116 µg/g of ash. Blood has shown the following lead distribution: erythrocytes, 24 µg/100 g, and plasma, 1.5 µg/100 g. Blood lead levels approaching 50 µg/100 g of blood warrant therapy in order to avoid the central nervous system (CNS) syndrome of adult or pediatric plumbism. Lead is normally found in hair in higher concentrations (on a weight-per-weight basis) than in any other tissue. The determination of lead in scalp hair is a valuable diagnostic aid in chronic or mild intoxication using atomic-absorption spectroscopy. In the distribution of lead in fetal tissues, lead seems to pass from the mother through the placenta to the fetus at the expense of blood.

The average individual is in lead balance, and the body burden of lead is established early in life and does not change appreciably through the life span. Feces and urine excretions, perspiration, and miscellaneous processes, in decreasing order, help to maintain balance. Lead is primarily excreted by the bowel as unabsorbed lead (90 percent), with urinary excretion being about 10 percent of the amount ingested. The amount of lead in the feces is practically equivalent to that obtained through food and lead intake, plus the unresolved portion secreted into the alimentary tract in the biliary digestive and mucous secretions.

Both acute and chronic lead poisoning have been recognized in children and adults. Acute intoxication results from the ingestion of metallic lead, soluble lead compounds, and lipophylic compounds (TML and TEL). Symptoms include leg cramps, muscle weakness, coma, impairment of renal function, and depression sometimes resulting in death. A syndrome identical with chronic intoxication may develop if sufficient lead is retained after acute plumbism. Chronic intoxication consists of three types: the gastrointestinal or abdominal, neuromuscular, and the CNS syndromes. They may occur separately or in combination. The neuromuscular and CNS syndromes result from intense exposure, whereas the abdominal syndrome tends to result from a slow and insidious developing intoxication. The CNS syndrome has been termed lead encephalopathy and is the most serious manifestation of lead poisoning in children. Blood-lead levels above 50 µg/100 g of blood and lead in hair of 42-975 µg lead per gram suggest chronic plumbism. It occurs rarely in adults and then only following massive exposure to lead fumes or TEL.

Interference by lead in the biosynthetic pathway for the formation of heme may result in encephalopathy. Diagnostic techniques for detecting lead intoxication in children include urinary coproporphyrin assay, serum delta-aminolevulinic acid (ALA) determination, lead assay of hair and whole blood, X-rays of bone, and urinalysis. Combinations of 2,3-dimercaptopropanol (BAL), ethylenediaminetetraacetic acid (EDTA), and edathamil calcium disodium are recommended for treatment of childhood plumbism. Plumbism in adults generally can be detected by blood and urine analyses, determination of delta-aminolevulinic acid, and urinary coproporphyrin assay. Blood-lead values above 80 µg/100 g and urine-lead values above 20 µg/100 ml are indicative of active plumbism and require immediate therapy. Adult plumbism, which results primarily from inorganic and TEL intoxication, is treated with EDTA-type chelating agents or penicillinamine.

An appraisal was made of the costs associated with acute lead poisoning of children to determine whether the effects of an environmental stressor upon man could be translated into pecuniary values. Cost estimates were developed for long-term treatment of patients suffering brain damage, as children from lead poisoning. The lifetime costs for care and treatment were estimated to be $200,000 to $250,000 for each individual, and the annual incidence of children suffering permanent brain damage in the United States was estimated at 1,900. Translation of effects of environmental stressors into monetary units appears feasible for stressors such as lead if there are acute exposures. No satisfactory method of handling costs associated with chronic exposures was found in this limited study.

A mathematical model was developed to represent the environmental transport of the stressor lead from several sources, with the subsequent intake of lead by man. In particular, the submodels for airborne transport of lead and the lead in the body were developed. The model developed for the airborne transport of lead was used to calculate the airborne concentrations of lead in the atmosphere due to emissions of lead from several sources. The predicted output is for an average steady-state airborne lead concentration, as the model was not developed to predict time-varying airborne lead concentrations.

The submodel, which represents the flow of lead into, within, and out of an individual, was developed in order to predict the distribution and quantity of lead in several body organs. Since measurement of the blood-lead level of an individual is a clinical method of investigating the effect of lead on an individual, the blood-lead levels due to several modes of intake of lead, including inhalation of various concentrations of airborne lead and ingestion of normal amounts and above normal amounts of lead, were calculated.

TECHNICAL, INTELLIGENCE, AND PROJECT INFORMATION SYSTEM FOR THE ENVIRONMENTAL HEALTH SERVICE
VOLUME III. LEAD MODEL CASE STUDY

by

Garson A. Lutz, Arthur A. Levin, Sanford G. Bloom,
Kaj L. Nielsen, Jack L. Cross, and David L. Morrison

INTRODUCTION

One of the charges given to the Environmental Health Service (EHS) is to establish a better understanding of the ecological system through consolidation of existing knowledge and acquisition of new knowledge. Under contract to the Environmental Health Service, Battelle Memorial Institute performed a study of the R&D planning processes in order to define a management assistance system for EHS that would permit efficient pursuit of this objective. Two concepts were central to the study – relating man to his total environment and planning within a framework of environmental stressors rather than categorical areas such as air, water, and solid wastes. Effective planning within the former concept requires the identification of all possible routes through the environment that a stressor can follow to reach man, an assessment of the relative importance of each path, and the prediction of the impact of the stressor upon man. By focusing upon environmental stressors, attention is continually directed at cause-effect relationships, material balances can be performed, and the specifics of removal of causes or treatment of effects can be handled within a single framework.

To aid in the development of the management assistance system for EHS, two model case studies were performed concurrent with the system developmental efforts. The environmental stressors – lead and persistent pesticides – were selected by mutual agreement between EHS and Battelle. These case studies were to provide specific examples for the development and evaluation of concepts during the project, especially for those tasks directed at the information network analysis and the development of mathematical models. The lead model case study is discussed in this volume and the pesticide study is discussed in Volume IV.

Lead and its compounds find extensive use in all sectors of man's environment. Lead is not essential to the nutrition of humans or animals and it is a heavy metal that does not have some modern therapeutic application. The acute exposure to lead produces severe effects in man, and chronic exposures within our present environment are undesirable. Since an extensive information base exists for lead, and since lead can reach man through several environmental pathways, it was selected as the stressor for a model case study.

OBJECTIVES

The utility of the R&D, Program Management, and Planning Assistance capability was demonstrated by applying this system to the development of management information on specific environmental stressors. All elements of the management-assistance system, which includes priority rating and R&D planning, the stressor matrix, the mathematical model, and the information network, were exercised through model case studies on lead as an environmental stressor. The objective of this model case study was to determine how effectively the management-assistance system can function as structured during the project, pinpoint areas where improvement of the system is needed, provide examples of the types of information on the environmental stressor (lead) that are currently available, and to indicate in a preliminary manner the additional R&D and other projects that should be considered by EHS in light of the present situation. A rating of the lead stressor was developed in order to suggest priorities for elements within the problem areas, and mathematical models were developed to investigate alternative approaches to the problem areas and to identify the most sensitive factors within the areas.

INFORMATION BASE

The lead literature is both diverse and of sufficient depth to provide a thorough evaluation of the information base that must be considered by EHS and the opportunity to test the validity of the environmental stressor concept. Examination of published data suggested that the basic information needs for the lead case study would be data on the production, utilization, ultimate disposal, and plumbism of lead and lead compounds. Three sources of information were selected, and appropriate categories in each identified. These are (1) environmental monitoring systems, (2) centers of environmental health information, and (3) published literature (newsletters, periodicals, abstract journals, and trade association releases). These three sources provided answers concerning:

(1) Lead Mines and Reclamation Sites. Location, employers, production, number of employees, technologies, absenteeism, monitoring data (mine, community), health patterns (mine, community), and toxicological data, including mutagenesis.

(2) Lead Compound Production. Identification, production, manufacturing or fabrication sites, number of employees, technologies, absenteeism, monitoring data, health patterns, and toxicological data. Also shifts in total production, in relative use of patterns, and possible effects of these shifts upon public health.

(3) Lead and Lead Compound Use Patterns. Identification of uses in production, manufacturing or fabrication of other consumer products, use of these products, and possible effects of use upon public health.

(4) Specialty Lead Compounds, Potentially New Lead Components. Delineation of selected categories from (1) and (2) above.

Occurrence of Lead(1)*

Lead occurs in nature principally in the form of sulfide ores (galena, PbS) or oxide or carbonate ores (anglesite, $PbSO_4$, and cerussite, $PbCO_3$). Minerals of iron, zinc, silver, copper, gold, cadmium, antimony, arsenic, and bismuth may be associated in varying proportions in lead ore deposits.

Production of Lead(2)

Lead is mined throughout the world. In 1968 production was about 3,500,000 tons. In addition, about 1,000,000 tons are recovered annually from secondary or scrap sources. Major mining areas yielding over 100,000 tons a year are the United States, Canada, Mexico, Peru, Yugoslavia, Australia, the USSR, Bulgaria, and Red China.

Mining, smelting and refining, secondary recovery, and imports are the four main sources of lead in this country. Tables 1 and 2 and Figure 1 illustrate the location of U. S. lead production mines, smelters, and refineries. During 1968, these mines produced approximately 359,000 short tons of lead, with Missouri, Idaho, and Utah accounting for 68 percent of the domestic output. Excluding on-site office workers, 8,700 men providing 18,330,000 man-hours were engaged in mining lead/zinc, of which 3,500 men provided 7,332,000 man-hours for mining lead.

Six companies (Table 3) are credited with the approximately 600,000 tons of lead produced in 1968 and 1969 by refining and primary smelting. The primary smelting and refining lead industry includes establishments engaged in smelting lead from the ore, and in refining lead by any process. Excluding on-site office workers, 2,400 men providing 5,545,000 man-hours were engaged in 1968 in on-site smelting and refining operations. Since approximately 20 percent of the lead obtained by smelting and refining was processed from imported ores, the manpower requirements might have been proportionately higher if operations were not conducted at a mining site.

Lead (as metal) imported in 1968 amounted to 338,120 short tons. This, added to the lead produced at primary refineries and secondary smelters in the U. S. gave a total new supply of approximately 1.39 million short tons.

In addition to the primary sources of lead, 550,879 tons of lead were obtained in 1968 (Table 4) from secondary recovery.

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*References are given on page 84.

TABLE 1. PRIMARY-LEAD PRODUCTION AREAS IN THE UNITED STATES

State		District	County or Counties	1968 Production, short tons
Arizona	1[a]	Glove	Santa Cruz	1,704
California	2	Death Valley	Inyo	4,001
	3	Death Valley	Inyo	
Colorado[b]	4	Creede	Mineral	19,778
	5	Ouray	Ouray	
	6	Butte	Gunnison	
	7	Gilman	Eagle	
	8	Rico	Dolores	
	9	Silverton	San Juan	
Idaho	10	Page	Shoshone	54,790
	11	Kellogg	Shoshone	
	12	Clayton	Custer	
	13	Wallace	Shoshone	
	14	Mullan	Shoshone	
	15	Osburn	Shoshone	
	16	Burke	Shoshone	
	17	8 Miles West of Hailey	Blaine	
Illinois	18	Rosiclare	Rosiclare	1,467
	19	Rosiclare	Rosiclare	
Kentucky	20	Marion	Marion	--
Missouri	21	Higdon	Perry	212,611
	22	Magmont	Bent	
	23	Ellington	Reynolds	
	24	Flat River	St. Genevieve	
	25	Bonne Terre	Iron	
	26	Bonne Terre	Reynolds	
	27	Bonne Terre	Washington	
Montana	28	Butte	Silver Bow	1,870
	29	Elliston	Powell	
	30	Boulder	Jefferson	
	31	Dillon	Beaverhead	
	32	Philipsburg	Granite	
Nevada	33	12 Miles from Gabbs	Nye	863
New Mexico	34	Vanadium	Grant	1,363
	35	Hanover	Grant	
	36	Bayard	Bayard	
New York	37	Balmat	St. Lawrence	1,396
Oklahoma	38	Gordon	Ottawa	2,387
	39	Tri-State	Ottawa	

TABLE 1. (Continued)

State		District	County or Counties	1968 Production, short tons
Utah	40	Mayflower	Wasatch	45,205
	41	Milford	Beaver	
	42	Bingham	Utah	
	43	Magna	Salt Lake	
	44	Ophir	Tooele	
	45	Lark	Salt Lake	
Virginia	46	Austinville	Wythe	3,573
	47	Ivanhoe	Wythe	
Washington	48	Metaline Falls	Pend Oreille	5,655
Wisconsin	49	Shullsburg	Lafayette	1,126
	50	Elmo	Grant	
	51	Mineral Point	Iowa	
Other States (Alaska, Kansas, Kentucky, Oregon)	52	--	--	1,367
Total				359,156

(a) Numbers designate areas in Figure 1.

(b) Keystone Mines Company recently announced a new plant located at Crested Butte, Colorado, for the production of silver, lead, and zinc concentrate-capacity 300 tons/day (Chemical Engineering, April, 1970, p 126).

TABLE 2. SMELTERS AND REFINERIES

State			County or Counties	S – Smelter R – Refinery
California	1[a]	Selby	Contra Costa	S & R
Idaho	2	Kellogg	Shoshone	S & R
Indiana	3	East Chicago	East Chicago	R
Missouri	4	Herculaneum	Jefferson	S & R
	5	Glover	Iron	S & R
	6	Bixby	Bixby	S & R
Montana	7	East Helena	Lewis and Clark	S
Nebraska	8	Omaha	Douglas	R
Texas	9	El Paso	El Paso	S
Utah	10	Tooele	Tooele	S

(a) Numbers designate areas in Figure 1.

TABLE 3. LEAD PRODUCTION BY SMELTING AND REFINING (S&R)

Company	Location of S & R Operations	1968 Production, short tons
Ascara (New Jersey)	East Helena, Montana	153,106
Bunker Hill (Delaware)	Kellogg, Idaho	124,134
International Smelting and Refining (Montana)	Perth Amboy, New Jersey	11,758
St. Joseph Lead (New York)	Herculaneum, Missouri	175,717
U. S. Smelting, Refining, and Mining (Maine)	Tooele, Utah (Toll basis)	35,486
Missouri Lead Operating Company (joint venture of American Metal Climax, Inc. and Homestake Mining Co.)	Bixby, Missouri	100,000
	Total	600,201

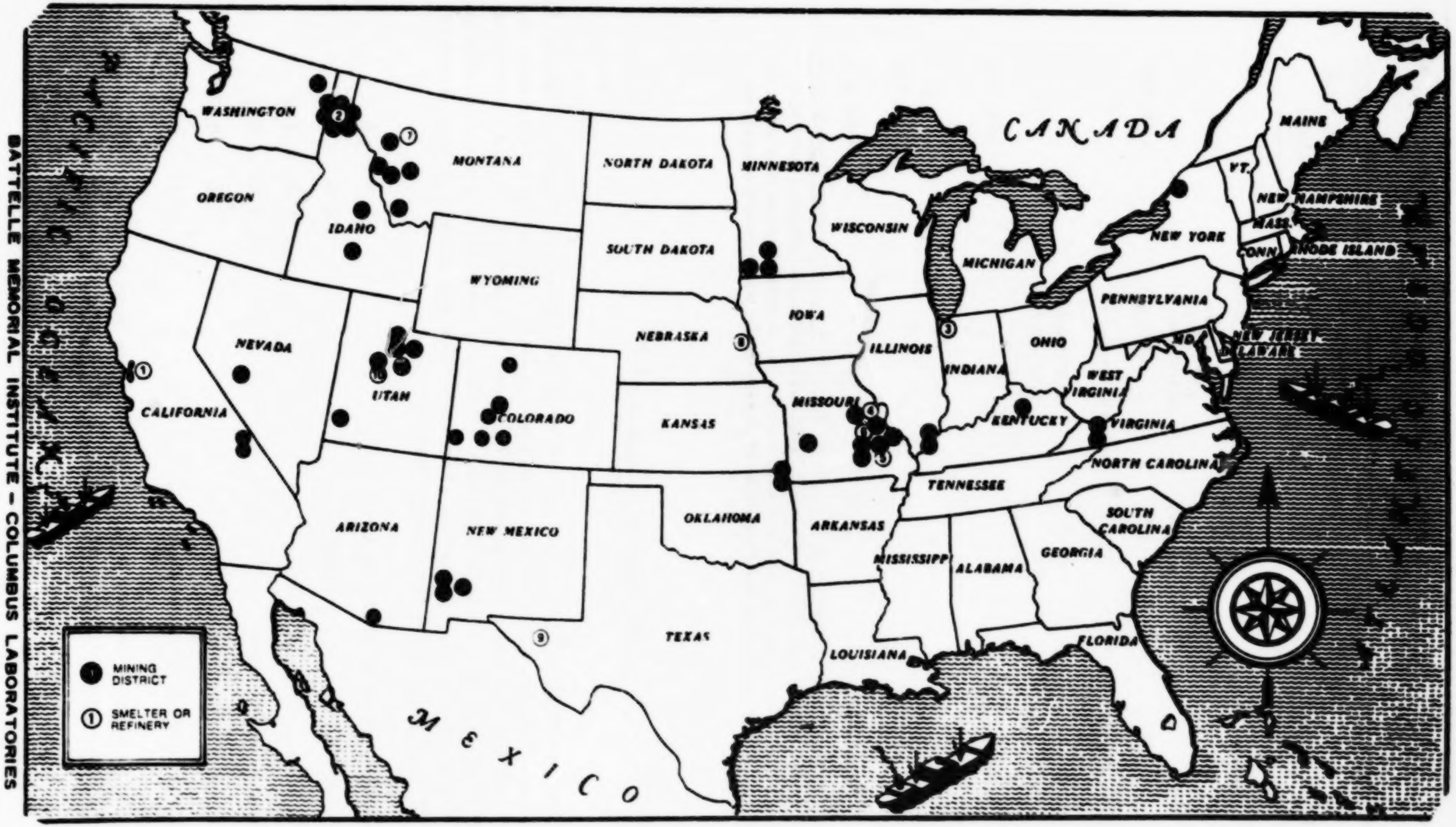
CANADA
MEXICO
PACIFIC OCEAN
ATLANTIC OCEAN
WASHINGTON
OREGON
CALIFORNIA
NEVADA
IDAHO
MONTANA
WYOMING
UTAH
COLORADO
ARIZONA
NEW MEXICO
NORTH DAKOTA
SOUTH DAKOTA
NEBRASKA
KANSAS
OKLAHOMA
TEXAS
MINNESOTA
IOWA
MISSOURI
ARKANSAS
LOUISIANA
WISCONSIN
ILLINOIS
MICHIGAN
INDIANA
OHIO
KENTUCKY
TENNESSEE
MISSISSIPPI
ALABAMA
GEORGIA
FLORIDA
SOUTH CAROLINA
NORTH CAROLINA
VIRGINIA
WEST VIRGINIA
MD
PENNSYLVANIA
NEW JERSEY
DELAWARE
NEW YORK
VT.
NEW HAMPSHIRE
MAINE
MASS
CONN
RHODE ISLAND
1
2
3
4
5
6
7
8
9
● MINING DISTRICT
① SMELTER OR REFINERY

FIGURE 1. PRIMARY-LEAD PRODUCTION AREAS IN THE UNITED STATES

Source: Lead Industries Association, Inc., New York, New York.

TABLE 4. SECONDARY LEAD RECOVERED IN THE UNITED STATES[a]

Kinds of Lead		1968 Production, short tons
As-refined metal		
At primary plants	2,259	
At other plants	136,607	
Total		138,866
In antimonial lead		
At primary plants	6,862	
At other plants	301,701	
Total		308,563
In other alloys	103,450	
Total		103,450
Grand Total		550,879

[a] 79,612 tons were from new scrap and 471,267 tons from old scrap. About 200 companies melt and smelt lead scrap. The total lead recovered from scrap materials has exceeded domestic mine production since 1945 and in many years has exceeded production of primary refined lead from both domestic and imported ores.

Upgrading of Lead Ores(1, 6)

Beneficiation or milling (Figure 2) is required of almost all lead ores except high-grade galena and oxide ores that may be amenable to direct smelting. The usual ore-dressing processes are crushing and grinding followed by flotation. Lead concentrate recovered from the float cells usually contains 45-60 percent lead (concentrated from the 3-8 percent of the mine rocks). Flotation chemicals commonly used in this process, and quantity in pounds per ton of ore treated are:

Sodium carbonate (conditioner)	1.0 – 2.0
Calcium oxide (conditioner)	2.0 – 4.0
Copper sulfate (activator)	0.8 – 1.2
Xanthate (collector)	0.01 – 0.2
Pine oil cresylic acid (frothers)	0.2 – 0.2
Sodium cyanide (repressant)	0.1 – 0.3

This 45-60 percent lead concentrate will contain varying percentages of zinc, silver, copper, arsenic, and other minerals originally in the mined crude ore.

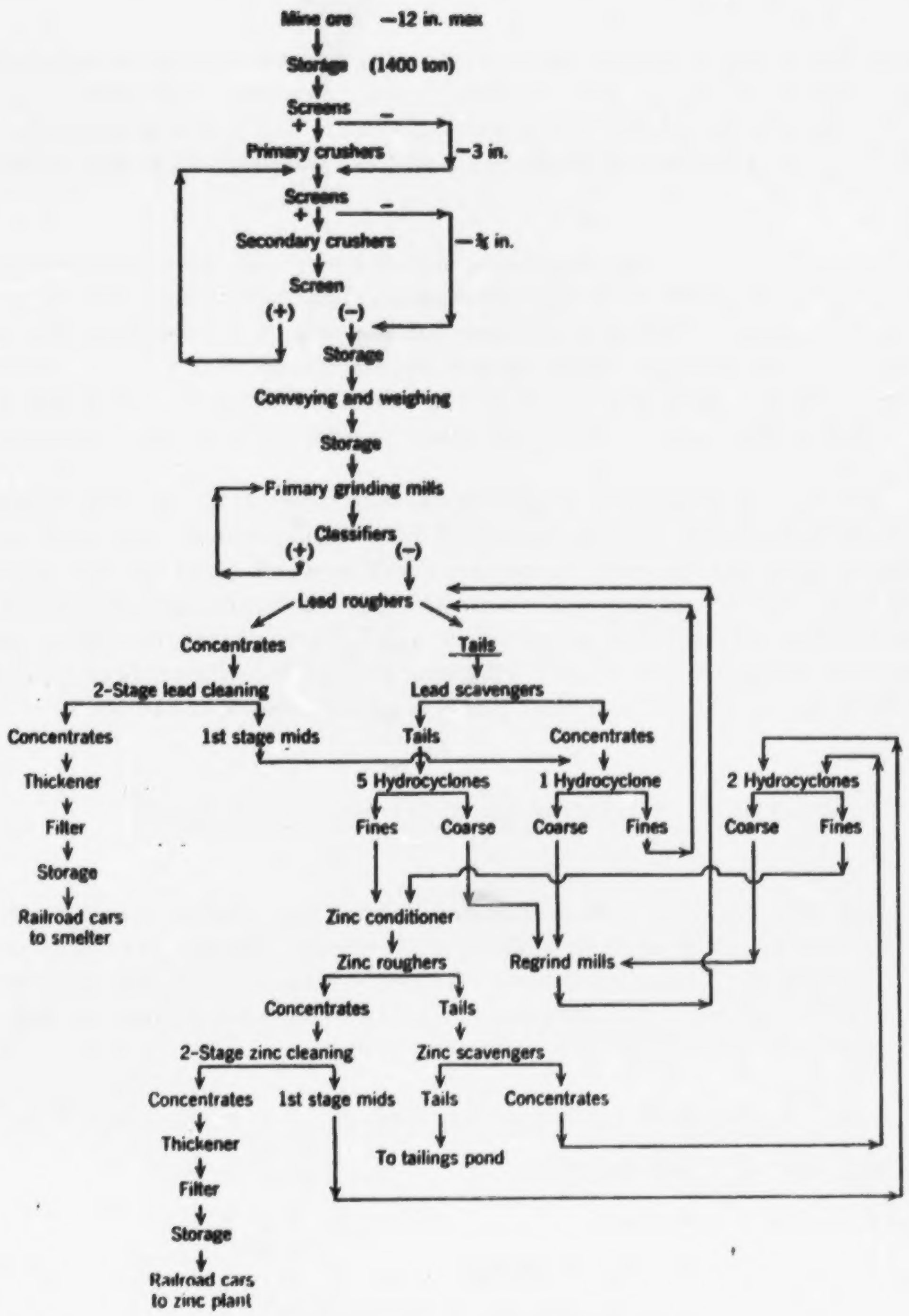
Mine ore —12 in. max
Storage (1400 ton)
Screens
+
—
Primary crushers
—3 in.
Screens
+
—
Secondary crushers
—⅜ in.
Screen
(+) (—)
Storage
Conveying and weighing
Storage
Primary grinding mills
Classifiers
(+) (—)
Lead roughers
Concentrates
Tails
2-Stage lead cleaning
Lead scavengers
Concentrates
1st stage mids
Tails
Concentrates
Thickener
Filter
Storage
Railroad cars to smelter
5 Hydrocyclones
1 Hydrocyclone
2 Hydrocyclones
Fines
Coarse
Coarse
Fines
Coarse
Fines
Zinc conditioner
Zinc roughers
Regrind mills
Concentrates
Tails
2-Stage zinc cleaning
Zinc scavengers
Concentrates
1st stage mids
Tails
Concentrates
Thickener
To tailings pond
Filter
Storage
Railroad cars to zinc plant

FIGURE 2. MILLING/BENEFICIATION OF LEAD ORE

Source: Kirk-Othmer Encyclopedia of Chemical Technology, Second Edition, Interscience Publishers, New York (1967), "Lead" (J. E. McKay), p 211.

After concentrating and pressure leaching (if practiced) the ore, the next steps are sintering (sulfur elimination) charge preparation, and the reduction of the lead compounds to the metallic form (bullion). This pyrometallurgical operations is shown in Figure 3.

Pressure leaching (aqueous oxidation) of lead concentrates removes the major portion of the copper, zinc, cobalt, nickel, iron, arsenic, and antimony, which are a hindrance to presently known smelting procedures. This takes place in three steps: (1) oxidation, (2) liquid/solid separation, and (3) recovery of metals from leach solutions.

The pulp is charged to an autoclave which operates with a solution temperature of 400 to 500 F and at a pressure of approximately 700-900 psig. About 95 percent of the lead sulfide is converted to insoluble lead sulfate which remains in the autoclave residue, contaminated with silver, gold, some calcium and some iron. Ninety to 95 percent of the other mineral present are oxidized to soluble sulfates. The leach residue will contain 45 to 60 percent lead, depending upon the autoclave feed composition.

In July, 1968, the Missouri Lead Operating Company (a joint venture of American Metal Climax, Incorporated and Homestead Mining Company) started an operation at Bixby, Missouri, that is believed to be the first new smelter in the United States in 40 years. While using a conventional flow scheme, the Bixby unit features updraft sintering of lead concentrates, production of sulfuric acid from sinter machine gases, continuous tapping of the lead blast furnace, and vacuum dezincing of desilvered lead. Production of 100,000 tons/year of 99.99 percent pure lead has been achieved.

Industrial Utilization and Distribution of Lead(4, 7, 8)

Lead's unusual physical and chemical properties make it one of the most widely used nonferrous metals in metal products, pigments, chemicals, and miscellaneous applications. (Table 5) The automobile industry was the largest consumer of lead, taking about 59 percent (storage batteries and gasoline additives) of the 1968 consumption. Other important uses, by percentages, were

Red lead and litharge	6.8
Ammunition	6.2
Solder	5.3
Cable covering	4.
Caulking lead	3.7
All others	15.

Included in "all others" are 16 end uses separately classified that account for a total of approximately 200,000 short tons of lead.

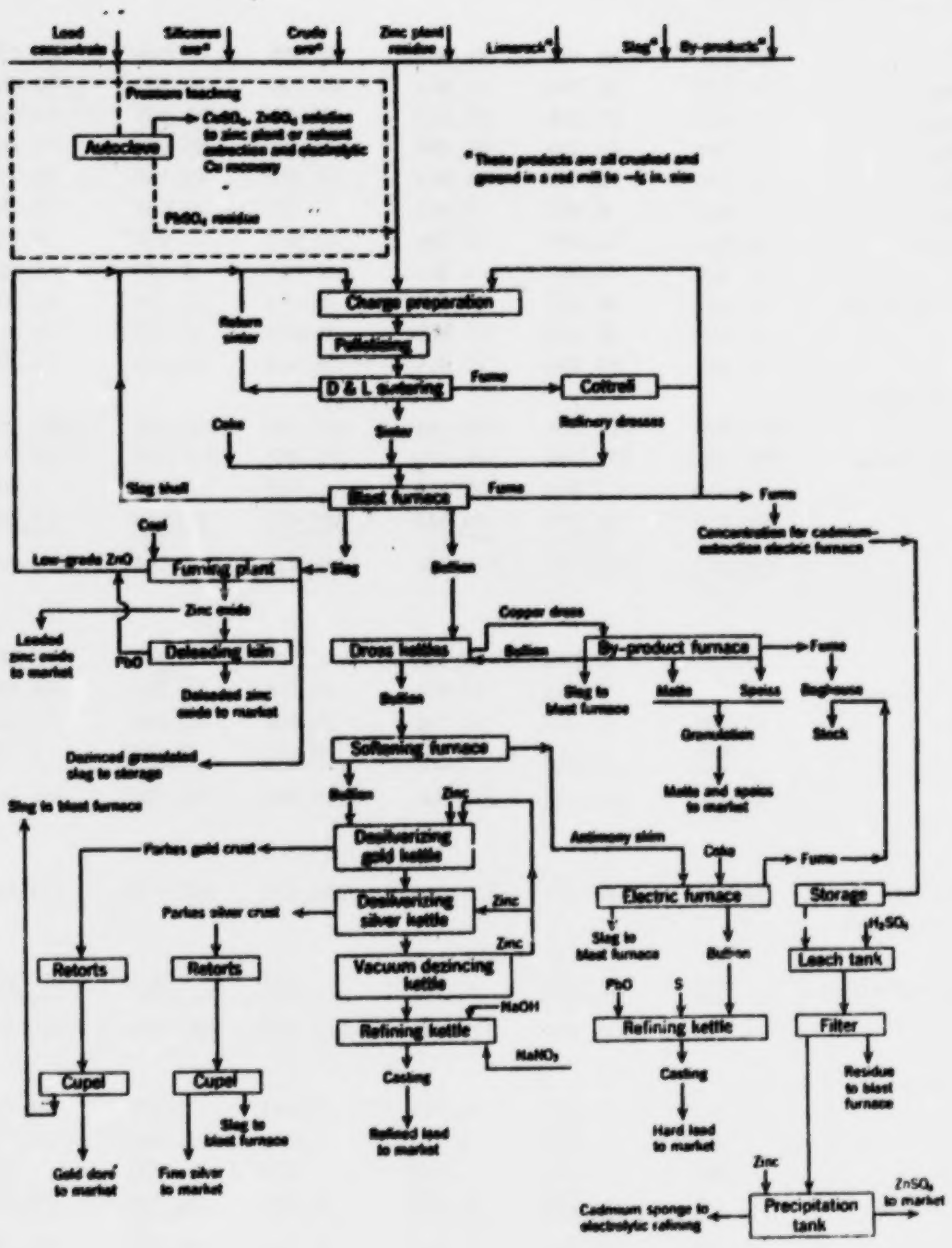
Lead concentrate
Siliceous ore*
Crude ore*
Zinc plant residue
Limerock*
Slag*
By-products*
Pressure leaching
Autoclave
CuSO₄, ZnSO₄ solution to zinc plant or solvent extraction and electrolytic Cu recovery
PbSO₄ residue
* These products are all crushed and ground in a rod mill to −⅛ in. size
Charge preparation
Return sinter
Pelletizing
D & L sintering
Fume
Cottrell
Coke
Sinter
Refinery drosses
Slag shell
Blast furnace
Fume
Fume
Concentration for cadmium-extraction electric furnace
Coal
Low-grade ZnO
Fuming plant
Slag
Bullion
Zinc oxide
Leaded zinc oxide to market
PbO
Deleading kiln
Deleaded zinc oxide to market
Copper dross
Dross kettles
Bullion
By-product furnace
Fume
Slag to blast furnace
Matte
Speiss
Baghouse
Granulation
Stack
Matte and speiss to market
Bullion
Softening furnace
Dezinced granulated slag to storage
Slag to blast furnace
Bullion
Zinc
Desilverizing gold kettle
Parkes gold crust
Antimony skim
Coke
Fume
Electric furnace
Storage
Desilverizing silver kettle
Zinc
Parkes silver crust
Zinc
Vacuum dezincing kettle
Slag to blast furnace
Bullion
H₂SO₄
Leach tank
Retorts
Retorts
NaOH
Refining kettle
NaNO₃
PbO
S
Refining kettle
Filter
Casting
Casting
Residue to blast furnace
Cupel
Cupel
Slag to blast furnace
Refined lead to market
Hard lead to market
Gold doré to market
Fine silver to market
Zinc
Cadmium sponge to electrolytic refining
Precipitation tank
ZnSO₄ to market

FIGURE 3. TYPICAL FLOWSHEET OF PYROMETALLURGICAL LEAD SMELTING

Source: Kirk-Othmer Encyclopedia of Chemical Technology, Second Edition, Interscience Publishers, New York (1967), "Lead" (J. E. McKay), p 215.

TABLE 5. LEAD CONSUMPTION IN THE UNITED STATES IN SHORT TONS

Products	Year						
	1963	1964	1965	1966	1967	1968	1969 (Preliminary Totals)
Metal Products							
Ammunition	49,804	56,403	57,322	78,435	78,766	82,193	77,805
Bearing metals	21,713	22,754	21,600	21,588	19,561	18,441	16,577
Brass and bronze	21,943	23,328	23,699	25,447	20,467	21,021	20,334
Cable covering	57,707	56,225	59,645	66,491	63,037	53,456	54,078
Caulking lead	76,308	73,628	66,584	63,250	48,789	49,718	41,888
Casting metals	7,856	6,961	5,046	6,671	10,083	8,693	6,482
Collapsible tubes	14,832	14,904	10,893	11,987	11,299	9,310	12,237
Foil	3,952	3,976	4,805	6,041	6,148	6,114	5,957
Pipes, traps, and bends	20,100	20,480	19,837	19,984	20,184	21,098	18,572
Sheet lead	26,495	29,605	27,569	28,938	26,763	28,271	22,675
Solder	67,945	71,186	77,819	78,898	68,833	74,074	65,126
Storage battery grids, posts, etc.	222,286	221,594	235,641	240,535	229,287	250,129	262,784
Storage battery oxides	216,795	207,754	219,706	231,957	237,378	263,574	295,337
Terne metal	1,983	1,609	2,109	1,966	1,620	1,427	1,563
Type metal	26,069	25,374	33,416	30,421	28,554	27,981	26,469
Total	835,878	835,871	865,691	912,609	870,769	915,500	927,884
Pigments							
White lead	8,846	8,802	8,414	8,131	8,087	5,857	6,617
Red lead and litharge	70,649	74,802	79,853	89,500	76,589	86,480	79,934
Pigment colors	11,767	11,921	12,553	13,695	13,041	14,163	14,670
Other[a]	7,813	8,111	8,063	8,562	5,473	3,234	665
Total	99,075	103,636	108,883	119,888	103,190	109,734	101,886
Chemicals							
Gasoline additives (primarily tetraethyl lead)	192,811	223,466	225,203	246,879	247,170	261,897	271,128
Miscellaneous chemicals	632	451	346	614	609	629	Not identified
Total	193,443	223,917	225,549	247,493	247,779	262,526	271,128
Miscellaneous Uses							
Annealing	4,847	5,699	5,719	5,441	4,202	4,194	2,353
Galvanizing	1,631	1,592	1,775	1,639	1,854	1,755	1,366
Lead plating	220	179	240	428	532	389	302
Weights and ballast	12,207	12,760	14,135	18,090	15,794	16,768	11,688
Total	18,905	20,230	21,869	25,598	22,382	23,106	15,709
Other Uses, Unclassified	16,057	18,484	19,490	18,289	16,396	17,924	16,596
Total Reported[b]	1,163,358	1,202,138	1,241,482	1,323,877	1,260,516	1,328,790	1,333,203
Estimated Undistributed Consumption	--	--	--	--	--	--	42,000
Grand Total	1,163,358	1,202,138	1,241,482	1,323,877	1,260,516	1,328,790	1,375,200

(a) Includes lead content of leaded zinc oxide production.

(b) Includes lead content of scrap used directly in fabricated products.

Tetraethyllead and Tetramethyllead(9, 10)

Lead consumption (Table 5) during 1968 in the manufacture of TEL and TML was about 525,000,000 pounds. The 1969 forecast for the manufacture of TEL and TML is 720,000,000 pounds, with a predicted growth rate of about three percent per year for the next 5 years.

Manufacturers and sites for the manufacture of TEL and/or TML are:

Ethyl Corporation	Baton Rouge, Louisiana Houston, Texas
E. I. du Pont de Nemours and Company, Inc.	Deepwater Point, New Jersey Antioch, California
Houston Chemical	Beaumont, Texas
Nalco Chemical	Freeport, Texas

Statistics on the number of production workers and tonnage production per TEL plant can only be approximated. For example, at its Baton Rouge plant, Ethyl Corporation has approximately 2100 employees in its Manufacturing Department in addition to sizeable numbers in Research and Development and Engineering Departments; at Houston approximately all of its 1000 employees are engaged in manufacturing. However, both plants manufacture several chemicals other than antiknock agents, e.g., sodium, hydrocarbons, and polyvinyl chloride). Also, a diversity of products is manufactured by Du Pont at its Deepwater Point plant. All factors considered, one chemical company estimates the value of TEL/TML sales at $400 million a year and that perhaps 6000 production workers are employed in making the two compounds in this country.

Storage Batteries(11, 12)

General statistics by geographical areas concerning the manufacture of storage batteries during 1967 are shown in Table 6. Accordingly, 233 establishments employing 15,800 production workers were engaged in the manufacture of storage batteries scattered throughout the country.

An inspection of the materials requirements and manufacturing processes for the lead-acid storage batteries, e.g., preparation of pasted plates, preparation and assembly of grids, and particle-size distribution of and grinding of lead oxides, suggests that lead poisoning might be anticipated. Table 7 lists cases of lead intoxication found in 66 industries which came to the attention of the Division of Occupational Health of the Pennsylvania Department of Health from December, 1966, through July, 1967. The 66 industries employed a total of 16,688 employees. Of this number, 977 (5.86 percent) worked in areas in which there was a lead exposure. Of the 26 cases of alleged lead poisoning, 12 were received as workmen's compensation claims, 8 were discovered and reported by industrial hygienists as a result of routine inspections, and 6 were uncovered by industrial hygiene physicians while investigating other claims. Four cases were questionable because of the absence of a history of exposure and/or unequivocal laboratory studies. The frequency of symptoms (26 cases) was epigastric pain – 15, abdominal discomfort – 8, constipation – 8, vomiting – 7, fatigue – 7, nausea – 6,

TABLE 6. GENERAL STATISTICS BY GEOGRAPHIC AREAS ON THE MANUFACTURE OF STORAGE BATTERIES DURING 1967

Geographic Area	Establishments		All Employees		Production Workers			Value Added by Manufacture, million dollars	Cost of Materials, million dollars	Value of Shipments, million dollars	Capital Expenditures, New, million dollars
	Total	With 20 or more Employees	Number, 1,000	Payroll, million dollars	Number, 1,000	Man-Hours, millions	Wages, million dollars				
United States, total	233	120	19.3	133.3	15.8	30.6	97.7	260.6	314.3	579.5	16.2
New England	13	9	0.8	6.2	0.5	0.9	2.8	7.6	9.2	17.1	0.3
Middle Atlantic	29	16	4.9	35.8	3.5	7.2	24.4	61.1	64.7	126.8	4.1
East North Central	40	25	4.6	34.6	3.8	7.7	27.6	75.0	84.1	159.3	2.8
Illinois	16	10	1.4	9.2	1.1	2.1	6.7	24.3	28.0	52.9	0.8
West North Central	17	12	2.4	15.2	2.0	4.0	12.1	28.7	36.7	67.1	1.0
South Atlantic	36	18	2.3	13.7	1.8	3.6	9.3	26.6	34.6	60.9	3.4
Florida	13	5	0.8	4.5	0.5	1.1	2.6	7.3	8.5	15.6	0.5
East South Central	14	8	0.6	3.7	0.5	1.0	2.9	7.6	13.4	21.5	(a)
West South Central	26	8	1.1	7.2	0.9	1.8	5.4	15.9	21.8	38.5	0.7
Mountain	8	4	0.3	1.8	0.3	0.6	1.4	3.2	4.3	7.6	(a)
Pacific	50	20	2.1	15.1	1.8	3.6	11.6	34.8	45.7	80.6	(a)
California	37	16	1.[illegible]	13.5	1.6	3.2	10.4	31.4	40.4	71.8	1.7

(a) Individual figures withheld.

muscular aches - 3, sweating - 3, distress after eating - 2, weight loss - 2, and weakness - 2. The areas of exposure presenting the highest concentration as measured in the worker's breathing zone were the pourer's exposure room in the furnace areas, the mixing areas, frit application, grinder's areas, and spray painting. Other work areas with exposures to high concentrations were fuse filling, snagging operation, lead pot, buffing, and battery breaking.

TABLE 7. REPORTED LEAD POISONING CASES FROM DECEMBER, 1966, THROUGH JULY, 1967, IN PENNSYLVANIA INDUSTRIES

Industries Surveyed

- Farming
- Contracting
- Textiles
- Printing
- Chemicals
- Abrasives
- Glass
- Primary metals
- Metal fabrication
- Machinery
- Electrical
- Auto parts
- Stevedoring
- Scrap yards
- Laboratories
- Miscellaneous manufacturing

Cases by Type of Industry	
Battery manufacture	11
Battery breaking	1
Lead products	3
Scrap yard	1
Fan manufacturing and range hoods	1
Abrasive manufacturing	1
Demolition	2
TV-tube manufacturing	1
Printing-ink manufacturing	1
Brass foundry	1
Structural steel parts spray painting	1
Explosives	1
Steel mill	1
	1
Total	26

On assay (using a dropping mercury polarographic method) of 537 air samples taken in these areas, 188 were found to exceed the threshold limit value (TLV for lead) of 0.2 mg/m^3 of air determined for an 8-hour day for a total exposure of 40 hours per week. In 39 plants where the air-sample concentrations exceeded the TLV, a standard medical recommendation was prescribed. Thus, if an employee showed a concentration greater than 0.15 mg of lead per liter of urine, a medical evaluation including a blood analysis was necessary. If the blood-lead concentration exceeded 0.10 mg lead per 100 grams of blood, the employee was removed from the lead exposure area until urine and blood levels of lead did not exceed the above specifications.

Nonferrous Foundry(14)

In a nonferrous foundry employing 75 workers, monitoring of the lead concentrations in the melting room, pouring room, and skimming area was initiated. An increase in concentration was noted over the years and found to be due to increased lead in the alloys and to the development of some poor work habits. Installation of mechanically exhausted enclosures for melting stations and movable, natural-gravity hoods and skylight fans over the skimming area and pouring area reduced the concentration of atmospheric lead from >0.15 mg lead/m^3 to 0.04 mg lead/m^3 (dithizone method). Good correlation was found between increased lead absorption, coproporphyrin excretion, and urine-lead levels.

Shipbuilding and Shipscrapping(15)

In the United States about 100 million tons of scrap iron are used annually in the production of about 100 million tons of pig iron and about 150 million tons of steel. A substantial portion of this scrap-metal tonnage is obtained from the "secondary metals" industry, including lead-based alloys. Metal burning is done constantly in some communities. During 1968, 86,500 tons of red lead and litharge were used to provide a protective coating for water tanks, bridges, buildings, and ships. In order to weld on these iron surfaces, temperatures exceeding 3000 C must be attained. Under these conditions numerous toxicants, including lead, are readily volatilized and various forms of metal-fume fever and intoxication can result.

In summary, lead intoxication seems to be occurring regularly but its symptons frequently are missed in the shipbuilding and shipscrapping industries. Genuine diagnosis and treatment await intensified instruction of employers, workers, and physicians. Health and safety codes must be tightened and enforced, including mandatory regular medical checkups. Diagnostic techniques must be improved and public instructional media utilized. Close and continuing teamwork must be generated between health and safety officials, private doctors, insurance carriers, employers, and workers.

Lead-Contaminated Whiskey(16, 17)

Toxic lead exposure in young women has a devastating effect on reproduction and pregnancy, since it frequently causes sterility or early spontaneous abortion. Lead crosses the human placenta and may cause untold effects in the fetus. Observations by Paul A. Palmisano, M. D., and his staff at the University of Alabama, Birmingham, Alabama, showed that three maldeveloped babies with similar symptoms had been born of alcoholic mothers who were moonshine drinkers. One infant that was examined in detail had intrauterine growth retardation and postnatal failure to thrive, combined with spastic and other nervous-system disturbances. The gravity of the possibility of maldevelopment of infants is realized when it is recognized that more than 17,000 moonshine stills were seized in the southeastern United States in 1966. A recent survey based on chemical analysis of untaxed whiskey samples disclosed that 53 percent contained concentrations of lead above 1000 micrograms per liter. According to Dr. Palmisano, during a recent 16-month period, 26 nonpregnant adult women admitted to the University of Alabama Medical Center showed symptoms of lead poisoning analogous to those obtained from the chronic ingestion of moonshine.

Cookware(18, 19)

Mexican pottery and earthenware for cooking have recently been identified as possible sources of lead poisoning. The lead is leached into the food. Recent FDA tests showed that 26 of 28 Mexican cookware samples were coated with glazes that contained lead. The FDA began its investigation after a doctor's family in the western United States contracted lead poisoning through continued use of such cookware.

Environmental Pathways for Lead Compounds(20)

An examination of the lead industry suggests that the adverse (physiological and psychological) effects of lead on man may be exercised through various transport paths, e.g., air, water, soil, and food. For example, mining, upgrading, and reclamation might be expected to contribute to atmospheric contamination which could affect adversely both the personnel engaged in these operations and the general populace living in the processing area. The automotive combustion products of leaded gasolines are transported to man through the atmosphere or food and may lead to adverse physiological effects. Paint formulations and painted surfaces containing lead compounds might be a hazard on contact or ingestion, and lead to pediatric plumbism. In order to perceive the flow of lead and its compounds through the environment, Table 8 was formulated as a reference base.

BIOCHEMISTRY AND TOXICOLOGY

Background

Lead is the only heavy metal that does not have some modern therapeutic application and is not essential to the nutrition of animals or human beings. The modern interest in the physiological properties of the metal centers around its toxicological interests.(21)

Gilfillan's explanation, derived from toxicology, vital statistics, archaeology, bone assays, and old Roman recipes and lead-lined pots for brewing poisons, suggests that plumbism was a major factor in the decline and collapse of the Roman Empire.(22) Lead poisoning of the wives and mistresses of upper class Romans occurred chiefly through their diet after the introduction of Greek cookery around 150 B.C. The most significant sources of lead were wine, grape syrup, and preserved fruit. Lead in women produces sterility, miscarriage, stillbirth, heavy child mortality, and encephalitis. Thus plumbism literally wiped out the middle and upper classes while the Roman free-poor and the slaves multiplied because they had much less lead in their diet.

The toxic action of lead was recognized by Hippocrates, in 370 B.C., who reported a severe attack of colic in a person engaged in extractive metallurgy. In 200 B.C., Nicander related constipation, abdominal pain, and pallor to the presence of this element in the body. By the first century A.D., lead poisoning was a well-recognized clinical entity, and Dioscorides fully described the syndrome. However, it was not until the 7th century that the more subtle forms of lead poisoning were observed. The first record of experimental investigations on lead is found in a treatise on toxicology by Orfila (1814). In 1831, Thackrah first definitely associated lead with the ill health of miners. However, lead mining itself has never been associated with the most severe cases of lead poisoning. Important areas in the United States, where exposure must be

TABLE 8. ENVIRONMENTAL PATHWAYS FOR LEAD COMPOUNDS

Chemical Compounds	Source	Transport Path	Effect	Uses
Lead acetate	Industrial processing	Air Water Land	Physiological	Dyeing and printing cottons Manufacture of lead salts Astringent (veterinary) Paint drier Lead coating of metals
Lead alloys	Industrial processing Power generation Transportation	Air Water Land Fisheries	Physiological	Slush coatings Type metals for printing Batteries Battery grids Bearing metals Solders Fusible alloys Communication cables Pipe and sheet in chemical installations Corrosion protection coatings
Lead antimonate	Industrial processing	Air Water Land		Pigment Staining glass Crockery and procelain
Lead arsenate	Industrial processing Agriculture	Air Water Land Food Consumer products	Physiological	Insecticides Veterinary medicine
Lead arsenite	Industrial processing Agriculture	Air Water Land Food Consumer products	Physiological	--
Lead azide	Industrial processing	Air Water Land	Physiological	Primer in explosives
Lead borate	Industrial processing	Air Water Land	Physiological	Paint drier and production of conductive coatings Glass Pottery Porcelain Chinaware
Lead carbonate	Industrial processing	Air Water Land	Physiological	Paints Ceramics Glazes Processing of parchment

TABLE 8. (Continued)

Chemical Compounds	Source	Transport Path	Effect	Uses
Lead chloride	Industrial processing	Air Water Land Direct contact	Physiological	Manufacturing of white lead dyes Solder and flux
Lead chromate	Industrial processing	Air Water Land Direct contact	Physiological	Pigment Printing of fabrics Decorating china and porcelain
Lead cyanamid	Industrial processing	Air Water Land Direct contact	Physiological	Corrosion inhibitors (Antirust paints)
Lead dioxide	Industrial processing Mining	Air Water Land Direct contact	Physiological	Electrodes Dyes Rubber substitutes Manufacture pigments Analytical purposes
Lead fluorosilicate	Industrial processing	Air Water Land Contact	Physiological	Electrolyte in electrolytic refining of lead
Lead fumarate	Industrial processing	Air Water Land Direct contact	Physiological	Plastisols Phonograph records Electrical insulation
Lead iodide	Industrial processing	Air Water Land Direct contact	Physiological	Bronzing, printing Photography
Lead linoleate	Industrial processing	Air Water Land Direct contact	Physiological	Driers in paints
Lead molybdate	Industrial processing Mining	Air Water Land	Physiological	Paint Pigment
Lead monoxide, litharge	Industrial processing	Air Water Land Consumer service Contact	Physiological	Ointments Plasters Glaze Flux Pigments Analytical Driers

TABLE 8. (Continued)

Chemical Compounds	Sources	Transport Path	Effect	Uses
Lead naphthenate	Industrial processing	Air Water Land Direct contact Home environment Work environment	Physiological	Driers in paint
Lead nitrate	Industrial processing	Air Water Land Contact	Physiological	Match industry Pyrotechnics Chemical intermediates
Lead oleate	Industrial processing	Air Water Land Direct contact Home environment Work environment	Physiological	Driers in paint
Lead orthophosphate	Industrial processing	Air Water Land Home environment Work environment Neighborhood environment School environment Consumer products Food Waste	Physiological	Stabilizer for plastics Pigments Anticorrosive agents
Lead phosphate	Industrial processing	Air Water Land Home environment Work environment Neighborhood environment School environment Consumer products Food Waste	Physiological	Stabilizer for plastics
Lead selenide	Industrial processing Mining	Air Water Lead Environment	Physiological	Semiconductor Infrared detector
Lead silicate	Industrial processing	Air Water Land Environment	Physiological	Glass ceramics
Lead stannate	Industrial processing	Air Water Land Environmental contact	Physiological	Ceramics

TABLE 8. (Continued)

Chemical Compounds	Sources	Transport Path	Effect	Uses
Lead subacetate	Industrial processing	Air Water Land Food	Physiological	Sugar analysis
Lead sulfate	Industrial processing Mining	Air Water Land Contact	Physiological	Pigments Batteries Lithography Fabrics
Lead sulfide	Mining Industrial processing	Air Water Land Contact	Physiological	Photoelectric cell Infrared detector Photosensitive resistor circuits
Lead telluride	Mining Industrial processing	Air Water Land Contact	Physiological	Photoelectric cell Infrared detector Photosensitive resistor circuits
Lead tetraoxide, red lead	Industrial processing	Air Water Land	Physiological	High voltage lightning arrested Dyes Chemicals Matches Pyrotechnics Curing agents (rubber substitute)
Lead tetraethyl	Industrial processing Power generation	Air Water Land Direct contact Food	Physiological	Antiknock Fungicide
Lead tetramethyl	Industrial processing Power generation Transportation Agriculture	Air Water Land Direct contact Food	Physiological	Antiknock Fungicide Filling of Geiger counters
Trialkyl or alkyl aryl lead	Industrial processing	Air Water Land Direct contact Food	Physiological	Polymerization Catalysts Stabilizers for PVCl Electroplating Lubricating oils Biocidal agents
Lead thiocyanate	Industrial processing	Air Water Land Contact	Physiological	Primer in explosives Matches Dyes
Lead thiosulfate	Industrial processing	Air Water Land Contact	Physiological	Rubber accelerator Lead mirrors

TABLE 8. (Continued)

Chemical Compounds	Source	Transport Path	Effect	Uses
Lead titanate	Mining Industrial processing	Air Water Land Contact	Physiological	Pigment Ceramics
Lead tungstate	Mining Industrial processing	Air Water Land	Physiological	Pigment
Lead vanadate	Industrial processing	Air Water Land Contact	Physiological	Pigment
Lead zirconate	Industrial processing	Air Water Land	Physiological	Pigment

guarded against, are the smelting, nonferrous alloy, battery, paint, ceramics, printing and plastics, and lead alkyl (gasoline additives) industries.(21)

Drinking of moonshine whiskey distilled by use of an automobile radiator has resulted in epidemics of acute lead poisoning among adults, including pregnant women.(16, 17) Lead-containing ceramic pitchers and in a few instances contaminated drinking water have served as a gastrointestinal source of lead intoxication.(18, 19) Most of the lead which accumulates in man is ingested from the water he drinks and the food he eats. The average man ingests about 5 mg/yr from drinking water and 100 mg/yr from his diet. From respiration, he receives about 15 mg/year.

Most cases of industrial lead poisoning result from the inhalation of lead dust resulting from lead production and processing, natural lead fall-out, battery production, destruction and burning of leaded fuels, and manufacture of paints and pigments. Proper precautions in handling the antiknock compounds TEL and TML eliminate possible inhalation hazards and skin absorption. Means are available for controlling both the stack emissions and in-plant air supplies to prevent lead from entering the ambient air. Industrial processes (above) produce lead vapor, mist, or dust containing lead that exposes workers to inhalation and absorption of lead from respiratory-tract epithelium. Concentration of lead in ambient air may vary from less than 0.01 $\mu g/m^3$ in Thule, Alaska, to 4.0 $\mu g/m^3$ in Los Angeles, to 50 $\mu g/m^3$ along heavily traveled highways. The lead industry claims that workers breathing air containing 150 $\mu g/m^3$ lead for a 40-hour week show no ill effects over a working lifetime. Generally, health authorities agree that lead concentration in respired air should be below 150 $\mu g/m^3$ and TEL concentration should be below 100 $\mu g/m^3$, preferably 50 $\mu g/m^3$. Since particle size distribution of lead in ambient air is varied and only those particles 5 μ and smaller are inhaled, attempts to classify atmospheric exposure to lead by the amount of lead in the air can lead to false conclusions.

Distribution/Storage in the Body

The disbribution of lead in the body is of fundamental importance because it is a crucial factor in the development of lead plumbism. The studies by Kehoe(23), Schroeder(24), and many others(25), indicate that following absorption, inorganic lead is deposited in many tissues of the adult body. Concentrations in the soft tissue are low, ranging from about 20 μg per 100 grams in striated muscle to about 200 μg per 100 grams in liver; those in the skeleton are relatively high, ranging from about 400 μg to about 1200 μg per 100 grams in the ribs or vertebrae, to two or three times these levels in the femur. These concentrations are expressed in terms of the fresh or formalin-fixed tissues rather than in terms of the dry or ashed tissues.(26),(27)

The search for children with lead poisoning mainly due to eating lead-based paint led to the development of techniques for blood-lead determinations. Children with valu s between 40 to 60 μg lead per 100 ml blood are considered:

(1) Beginning ingesters whose lead concentration is increasing

(2) Previously poisoned children whose lead concentration is declining towards normal, i.e., 20 to 25 μg/100 ml in whole blood.(28, 29)

Lead is deposited in bone in the form of the insoluble tertiary lead phosphate. High phosphate intake favors skeletal storage of lead and a lowering of the lead content in soft tissues. Conversely low phosphate intake mobilizes lead from bone and raises the lead content in soft tissues. Vitamin D promotes the deposition of lead in bone if a sufficient amount of phosphate is available, otherwise deposition of calcium prevents deposition of lead. Parathyroid hormone and dehydrotachysterol mobilize lead from the skeleton and promote the blood-lead level and urinary excretion of the metal. Acidosis results in mobilization from bone and the excretion of both lead and calcium. Iodides and bicarbonates also favor movement of lead from bone. Lead in the bone salts does not contribute to toxicity.(21)

Teeth are confirmed to have a higher concentration of lead than any of the bones, and the average lead concentration increases with age, e.g., <10 years ca 17 μg/g of ash and 50 to 60 years ca 116 μg/g of ash.(24)

Lead is normally found in higher concentrations (on a weight-per-weight basis) in hair than in any tissue. The determination of lead in scalp hair is a valuable aid in diagnosing chronic or mild intoxication using atomic absorption spectroscopy. In a study of 93 presumably healthy children under 17 years of age from the greater Boston area, a mean led concentration of 31 μg/g of hair was found. In contrast, for 20 children under 8 years of age with chronic or acute plumbism the mean lead concentration was 276 μg/g of hair.(30)

The distribution of lead in fetal tissues and organs appears to follow that of the adult, i.e., in bones it tends to increase with their development. It is believed that lead passes from the mother through the placenta to the fetus at the expense of blood.(16, 17)

The distribution of TEL and TML in the tissues after absorption differs initially from that of inorganic lead, as do the final sites of deposition. Skin absorption of TEL in rabbits is followed by decomposition of TEL in tissue. In humans and in animals (rats), TEL and TML affect primarily the central nervous system. In rats the lead is stored in the blood, muscle, and pancreas after the administration of TML and TEL. Muscle of female rats contained more lead before than after the administration of either compound, while brain, liver, kidney, spleen, carcass, blood, and lung of males contained more lead than did the corresponding organs from females. After inhalation by rats, TEL can be recovered unchanged from the liver. Nevertheless, it seems to be gradually converted to inorganic lead so that chronic exposure may result in a toxic syndrome characteristic of that for inorganic lead. After dosage with TML, the blood of the rats contained more lead than did any tissue.(24, 31-32)

Excretion

The average individual is in lead balance, and the body burden of lead is established early in life and does not change appreciably through the life span. It is difficult to influence lead body burdens without resorting to drastic measures.

Feces and urine excretions, perspiration, and miscellaneous processes, in decreasing order, help to maintain lead balance. Lead is primarily excreted by the bowel with the greatest portion representing unabsorbed lead. The amount of lead in the feces is practically equivalent to that in food and beverages ingested. Roughly

90 percent of the ingested lead traverses the alimentary tract unabsorbed and is discharged in the feces. To this unabsorbed lead in transit is added the desorbed portion secreted into the alimentary tract in the biliary, digestive, and mucous secretions. The efficacy of fecal excretion of lead is reflected in the observation that ingestion of lead at a constant intake of 0.3 mg/day for 420 days produced no detectable elevation of blood lead. When the dose was increased to 1 mg/day for 4 years, the blood lead re reached only 46 μg/100 g, and doubling the dose gave a value of 65 μg/100 g after 2 years. Three milligrams daily, ten times the content in the daily diet for 4 months, produced a blood-level of only 50 μg/100 g.(26,33)

During lead balance, the urinary excretion of inorganic lead is about 10 percent of the amount ingested. The amounts excreted in the urine average 20 to 40 μg/l. After hazardous lead exposure, the amount of lead excreted is above 150 μg/l.(21)

It is not known whether there is a significant urinary excretion of TEL and TML, but there is essentially no correlation between the amount of organic lead inhaled and the lead concentration of urine. However, persons employed in the manufacture of TEL have been found to excrete abnormal quantities of lead.(21,31)

Types of Lead Poisoning

Lead poisoning is usually divided into acute and chronic forms. Although chronic poisoning correctly describes prolonged exposure, manifestations often are acute.

Acute intoxication may result from the ingestion of metallic lead but more commonly follows ingestion of a large amount of any soluble lead salt, especially the acetate, carbonate, or chromate. If absorption in the stomach is sufficient, pain, leg cramps, muscle weakness, paresthesias, depression, coma, and death may follow within 1 to 2 days. Although temporary disturbances in renal function may occur, lead is not recognized as a dangerous nephrotoxin. However, kidney damage may result in oliguria, albuminuria, and cylindruria. An acute hemolytic crisis sometimes develops and results in anemia and hemoglobinuria.(32-34)

Acute lead poisoning has followed exposure to TEL vapor. The pure liquid is capable of rapid penetration of the intact skin. As with many other heavy metals, the chief disturbance occurs within the central nervous system and manifests itself as insomnia, headache, restlessness, dizziness, irritability, ataxia, delusions, mania, and sometimes convulsions. The lack of proper controls combined with the use of TEL as a degreaser has in recent years resulted in a number of fatalities and intoxications in Yugoslavia and Italy. The toxicity of tetraalkyl leads appears to depend on their conversion in vivo to the trialkyl forms. Under these conditions, the urinary concentration of lead may be elevated above 150 μg/l. Continued absorption of small amounts of TEL can result in chronic lead poisoning since the lead is eventually converted in the body to inorganic lead.(21,26,32)

A syndrome identical with chronic intoxication may develop if sufficient lead is retained after acute lead poisoning. Fortunately, many acute poisonings subside without sequelae, since the absorption of lead from the bowel is slow and incomplete.

Chronic intoxication consists of three types: the gastrointestinal or abdominal, the neuromuscular, and the CNS syndromes. These may occur separately or in combination. The neuromuscular and CNS syndromes result from intense exposure to lead, whereas the abdominal syndrome tends to result from a slow and insidious developing intoxication. The abdominal type is observed principally among industrial workers and the CNS syndrome is more typical of pediatric plumbism, because of the persistent ingestion of lead-containing substances by children with pica.(33, 34)

The abdominal syndrome is characterized by anorexia, a metallic taste, constipation, and severe abdominal cramps (lead colic). The abdominal muscles become rigid, and tenderness in the region of the umbilicus is common. In adults, the body temperature may fall, but in children it usually rises. Pallor and elevated blood pressure have been observed. The neuromuscular type is characteristic of adult plumbism but has occasionally been reported in children. In the neuromuscular syndrome "lead palsy" weakness of a muscle group actually precedes paralysis. Extensor muscles of the upper extremities are more often paralyzed than flexors or lower extremity muscles. Wrist drop, and to a lesser extent, foot drop, are characteristics for this syndrome. The palsy is often unilateral, confined to the predominant side used, and may be limited to only one or two muscle groups. The extraocular muscles are almost frequently involved. Atrophy may occur after long-standing paralysis and may result in incomplete recovery of muscle function after termination of exposure.(21, 26, 35)

The CNS syndrome has been termed lead encephalopathy and is the most serious manifestation of lead poisoning in children. It occurs rarely in adults and then only following massive exposure to lead fumes or TEL in a salvage operation, or in cleaning large gasoline storage tanks. TEL is lipid-soluble so that exposure results almost exclusively in the encephalopathic form.(33-34)

Lead Poisoning in Children

Incidence

Childhood plumbism in the United States is largely a disease of the big cities and especially of the slum areas where old deteriorated housing prevails. The interior woodwork, painted plaster, and wallpaper of houses built prior to 1940 may contain layers of flaking lead-pigment paints which have never been removed. Several such flakes may contain far more than the average daily (0.3 mg) intake of lead. Thus, children with a history of pica are likely candidates for lead poisoning. In New York City, lead poisoning in children leads to death in about 15 to 20 percent of cases, and to neurologic and mental disturbances from encephalopathy in over 25 percent of the survivors. As shown in Table 9, of the more than 61,000 poisonings reported to the New York City Poison Control Center between 1954 and 1964, 3 percent or approximately 1,700 cases were due to lead. Deaths numbered 128. The apparent increased incidence of lead poisoning (comparison of 1964 with 1954) is believed due to the currently vigorous case-finding program, resulting in the discovery of cases in the asymptomatic state prior to the onset of encephalopathy. Encouraging is a sharp decrease in the number of deaths in recent years.(28, 29)

Jacobziner also found that 54 percent of all deaths from lead poisoning occurred in 2-year-old children. Lead encephalopathy is encountered much more frequently during the summer months but asymptomatic lead poisoning is a year-round disease.

TABLE 9. CASES OF CHILDHOOD LEAD POISONING PLUS FATALITIES IN NEW YORK CITY FROM 1954 to 1964

Year	Cases	Deaths	Case Fatality[(a)]
1954	80	12	15.0
1955	115	18	15.7
1956	99	9	9.1
1957	85	9	10.6
1958	116	21	18.1
1959	171	12	7.0
1960	146	18	12.3
1961	181	6	3.3
1962	198	9	4.5
1963	338	7	2.1
1964	509	7	1.4
Total	2,038	128	6.3

(a) Deaths per 100 cases.

However, 45 percent of the total cases reported between 1954 and 1964 occurred between June and September. The exact reason for this is not known. One postulation is that a metabolic disturbance increases lead absorption because of the actinic rays of the summer sun. Signs and symptoms of lead poisoning show some inconsistencies. In a random sample of 300 confirmed cases, 76 percent had no presenting complaints. Further inquiry and testing showed that 48 percent had anorexia, 9 percent had vomiting, 24 percent had roentgenologic changes in the long bones, but only 2 percent responded positively to X-ray of the abdomen. Twelve percent had encephalopathy. The blood-lead content ranged from 0.06 mg/100 g to 0.34 mg per 100 g with the average being 0.08 mg per 100 g of blood. Jacobziner considers the blood-lead determination as the most reliable procedure for identifying lead intoxication, particularly in asymptomatic cases. A blood-lead content of 0.06 mg/100 g of whole blood is considered abnormal (high) and warrants treatment.(36)

The Chicago Board of Health in October, 1966, began a mass screening program using a blood-lead test to detect lead poisoning in children living in substandard conditions. Atomic absorption spectroscopy made it possible to screen 5,000 specimens in 1 month and to test a total of 68,744 children in 1967 and 1968 (Table 10). Of the 68,744 children, 5.7 percent (3,935) had lead values of 50 μg (0.05 mg)/100 g of whole blood. Children with elevated lead values were referred to a special clinic established by the Chicago Board of Health for the diagnosis and treatment of lead poisoning. Twenty-nine percent (1,154) of these referrals were treated with chelates; the remainder were screened periodically until their lead level returned to normal. There were no deaths from lead poisoning among the 68,744 tested in 1967 and 1968. The incidence of high blood-lead levels among children living in the same areas declined from 8.5 percent in 1967 to 3.8 percent in 1968. Blood-lead values were lowest in November through January and highest in June.(37)

TABLE 10. DATA OF SURVEY AREAS FOR CHICAGO BLOOD-LEAD SCREENING PROGRAM - 1967-1968

		1967				1968				Totals for 1967-1968			
Urban Area	A Number of Children Under 5 Years (1960 Census)	B Number Tested(a)	Percent of A	Number With High Lead(b)	Percent of B	C Number Tested(a)	Percent of A	Number With High Lead(b)	Percent of C	D Number Tested(a)	Percent of A	Number With High Lead(b)	Percent of D
1	10,000	2,500	25	157	6.3	3,100	31	53	1.7	5,600	56	210	3.8
2	15,100	1,700	11	73	4.3	1,800	12	56	3.1	3,500	23	129	3.7
3	22,000	2,000	9	262	13.1	3,000	14	166	5.5	5,000	23	428	8.6
4	22,300	5,200	23	509	9.8	5,500	25	270	4.9	10,700	48	779	7.3
5	13,700	4,100	30	316	7.7	4,900	36	142	2.9	9,000	66	458	5.1
6	24,500	6,600	27	393	6.0	7,700	31	277	3.6	14,300	58	670	4.7
7	20,500	3,400	17	432	12.7	7,000	34	356	5.1	10,400	51	788	7.6
8	15,300	2,200	14	218	9.9	6,100	40	227	3.7	8,300	54	445	5.4
9	4,500	300	7	19	6.3	1,700	38	9	0.5	2,000	44	28	1.4
Totals	147,900(a)	28,000	19	2,379 =	8.5	40,800	28	1,556 =	3.8	68,800	47	3,935 =	5.7

(a) Figures given to the nearest hundred.
(b) High lead = 50 μg lead/100 ml whole blood or higher.

Table 11 summarizes the results of a survey conducted in Cleveland, Ohio, during the summer months of 1959 and 1960 on pica and environmental factors related to lead poisoning. Increases in concentration of both lead and coproporphyrin was observed in 216 (27 percent) of children from old housing areas. Plumbism was observed in 38 (4.7 percent). Background information showed that from 1952 through 1958, approximately 8 to 9 Cleveland children living in substandard housing were hospitalized per year for lead poisoning, with a mortality of 30 percent. (38)

TABLE 11. ENVIRONMENTAL EXPOSURE OF YOUNG CHILDREN TO LEAD IN URBAN HOUSING IN CLEVELAND

	Number of Children	Number with Abnormal Urine	Number with Plumbism
Old housing	801	216	38
New housing project	105	3	0

From 1956 through 1964, there were 564 clinically diagnosed cases of plumbism and 797 cases of abnormal lead absorption among Baltimore, Maryland, children or a total of 1,337 cases. The clinically diagnosed cases of lead poisoning were investigated as were cases in which a blood-lead level exceeding 0.06 mg/100 g of blood. Homes of patients were visited and painted surfaces accessible to the affected children assayed for lead. The owners were required to remove the paint from the indicated areas. An extension of this effort during 1957 showed lead in excess of 1 percent in paint used in 70 percent of 667 dwelling units. In the same year, assay of paint scrapings from 300 homes of indigent persons on medical care gave positive tests for lead in scrapings from 58 percent of the homes. (39)

Symptoms and Manifestations

The signs of lead poisoning are subtle, nonspecific, and insidious. Symptomatology includes anorexia, headache, vomiting, insomnia, anemia, visual disturbances, convulsions, hallucinations, and coma. Among preschool children lead poisoning should be considered when diagnosis indicates behavior disorders, convulsive disorders, and mental retardation. (21, 40-41)

Clinically and pathologically, pediatric plumbism manifests itself mainly in three systems: hematopoietic, gastrointestinal, and neurological. From the hematological viewpoint, the punctate basophilic stippling count of erythrocytes is only significant when considered with other signs of plumbism. More common than stippling is microcytic hypochromic anemia. Failure to respond to iron therapy would suggest the possibility of plumbism. (21, 26)

The gastrointestinal symptoms are difficult to relate exclusively to plumbism.

The most serious manifestations of pediatric plumbism are those involving the CNS. The biosynthetic pathway for the formation of heme is very sensitive to the toxic effects of lead with the result that the metabolism of delta aminolevulinic acid (ALA) coproporphyrin and protoporphyrin is disrupted. Thus, in plumbism, the levels of these metabolites in body fluids provide highly useful parameters of lead toxicity. ALA is increased in both plasma and urine. The urinary excretion of coproporphyrin is increased manifold. Free erythrocyte protoporphyrin is also increased. These derangements of syntheses lead to alterations in metabolic pathways and conceivably, brain hemodynamics.(26,33)

An electron microscopic study of biopsy specimens from children with lead encephalopathy indicated changes in the neurons in the gray matter and in the structure of the capillary endothelium. Changes in the white matter, glial cell nuclei, myelin sheath, and axons were noticed. These physiological changes probably contributed to the genesis of encephalopathic syndrome.(26)

Lead encephalopathy may produce early death, be followed by complete recovery or result in permanent sequelae. This cycle of events is representative of studies in many large cities east of the Mississippi River. For example, in a Chicago study, complete recovery occurred in 257 of 405 children. The remaining 168 had neurological sequelae distributed as follows: 93 showed mental retardation, 85 had recurrent seizures, 9 had cerebral palsy, and 5 developed optical atrophy. These cases of lead encephalopathy were produced by pica or inhalation of fumes from burning battery cases. Blood lead in most cases was 60 μg/100 g of whole blood or higher.(42)

Diagnosis

Table 12 lists the laboratory determinations compiled by Chisolm required for accurate diagnosis of childhood lead poisoning.(28,29) These tests consist of (1) emergency tests for rapid presumptive diagnosis and (2) specific diagnostic tests. Chisholm suggests that a battery of tests be employed. A positive history (especially if prolonged) calls for complete evaluation including blood lead hematocrit, hemoglobin, reticulocyte count, X-rays, and the urinary coproporphyrin tests or serum ALA determination. Evaluation of asymptomatic siblings or housemates of index cases calls for at least lead determinations and hematocrit. Elevated blood levels should be confirmed prior to treatment with chelating agents.

Therapy

Guidelines for the choice of appropriate chelation therapy also have been developed by Chisolm (Table 13).(28,29) The combination of BAL and EDTA, by providing sulfur as well as nitrogen and oxygen atoms, permits competition with a wide range of tissue ligands with which lead may be associated. The highest doses of chelating agents are required in those patients with the highest lead body burdens. Treatment of children with encephalopathy first with BAL and later with EDTA results in a fatality rate of 25 to 35 percent. This mortality can be reduced to 5 percent when BAL and EDTA are used together. However, it does not eliminate the occurrence of permanent brain damage among survivors. As shown in Table 13, a strong positive urinary coproporphyrin test or plasma ALA value >3 μg/ml suggests a high lead content and the need for immediate therapy with BAL and EDTA. When the blood lead lies between 60 and 80 μg of lead/100 g of blood, EDTA alone or d-penicillamine will give satisfactory results.

TABLE 12. LABORATORY DETERMINATIONS REQUIRED FOR DIAGNOSIS OF LEAD INTOXICATION IN CHILDREN[a]

Emergency Tests for Rapid Presumptive Diagnosis

Qualitative urinary coproporphyrin (UCP) test
  (intense orange-red fluorescence +++ or ++++ often assoc. with >80 μg Pb/100 g w. b.)

Serum δ-aminolevulinic acid (ALA)[b]
  Normal >0. 1 μg/ml
  Severe intoxication >0. 3 μg/ml

Lead in hair[c]
  Controls=2-95 μg Pb/g hair
  Chronic plumbism=42-975μg pb/g hair

X-rays
  Flat plate of abdomen
  PA view of wrists, knees

Blood Studies
  Hemoglobin, hematocrit, reticulocyte count (marrow smear preferred, if necessary, for basophilic stippling of normoblasts)

Urinalysis
  (+ to ++ glycosuria in very severe cases)

Specific Diagnostic Tests
(Lead-Free Collection Equipment Mandatory)

Whole-blood lead

Urine lead output
  (24 hr quantitative collection)
  Endogenous output – may be normal in acute encephalopathy

  Chelation therapy – output >1. 5 μg Pb/24 hr during 1st 24 hr of EDTA therapy, diagnostic of plumbism

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(a) Pediatrics, 44, 291 (1969).
(b) Pediat., 73, 1 (1968).
(c) N. Eng. J. Med., 276, 949 (1967).

TABLE 13. CHILDHOOD PLUMBISM

(Guidelines for Choice of
Chelating Agents)

	Diagnostic Criteria				
Laboratory Data					
Blood Lead µg Pb/100 g w. b.	< 100	80-100	60-80	40-60	40-60
Urine coproporphyrin (qualitative test)	++++	+++	0 to ++	++	0
Plasma ALA, µg/ml	>0.30	>0.25	>0.20	>0.20	>0.20
EDTA mobilization, µg Pb/24 hr	Test unnecessary			>1000	>600
Clinical Status					
Encephalopathy	+	±	0	0	0
Intoxication	+	+	±	+	0
Asymptomatic	+	+	+	+	+
Therapy(a)					
Immediate (in hospital)	BAL-EDTA(b, c) 5-7 days	BAL-EDTA 3-5 days	d-pen(d) (EDTA) 5 days	d-pen 5 days	0
Long term	d-pen	d-pen	d-pen	±	0
(in convalescent facility or safe home)	6-12 months		2-6 months	±	0

(a) Selection of drugs and dosage based mainly on blood lead concentration.

(b) BAL = 2, 3-dimercaptopropanol.

(c) EDTA = Edathamil calcium disodium.

(d) d-pen. = d-penicillamine.

Where blood lead lies between 40 and 60 μg/100 g of blood, therapy is not needed unless metabolic evidence warrants.

Adult Plumbism

Plumbism in the adult generally can be traced to poor industrial hygienic practices in the industrial use of inorganic and organic lead salts, smeltering and refining operations, and in the manufacture of batteries or accessories.

Symptoms and Manifestations

Adult or chronic plumbism requires months and sometimes years to develop and depends both upon increased exposure and absorption of lead until chronic intoxication is induced. Symptoms are similar to those described for chronic intoxication and types of lead poisoning. General physical impairment, intestinal disturbances, hematological alterations, nephropathy, and neuromuscular paralysis have been reported. Toxic lead exposure of young women has a devastating effect on reproduction and pregnancy since it most commonly causes sterility or miscarriage. Excessive lead in the water supply of pregnant women has been reported to result in 7 miscarriages out of 8 pregnancies. Neurologic defects, intrauterine-growth retardation, and postnatal failure to thrive have been observed in an infant whose mother during pregnancy had ingested lead contaminated whiskey. No cases have been reported to indicate that the amount of lead in the air has any effect on pregnancy.(1, 16, 23)

Diagnosis

Although many tests have been investigated, the following are considered the most valuable for detecting lead intoxication.

(1) Determination in blood and urine levels employing spectrophotometric, spectrographic, polarographic or atomic absorption procedures. Blood lead values above 80 μg/100 g and urinary lead values above 200 μg/l are indicative of active plumbism and require immediate therapy.

(2) Determination of δ-aminolevulinic acid (ALA). By its interference with the heme synthesis at the ALA step, lead causes an increased excretion of ALA in plumbism, including latent plumbism. Increased urinary ALA has been demonstrated for periods up to 3 years after the last known exposure to lead. Normally 2 mg of ALA or less are excreted per 24 hours. Lead poisoning results in a 20 to 200-fold increase in excretion.

(3) Urinary coproporphyrin test. This is a widely used indicator of lead exposure. The test should be confirmed by urine and blood lead analyses and aided by clinical examinations. Normally 60 to 280 μg of coproporphyrin are excreted per 24 hours. Lead poisoning results in a three- to tenfold increase in coproporphyrin excretion.

(4) Edetate lead mobilization test coupled with the determination of both ALA and coproporphyrin. After administration of the chelate to lead-poisoning victims, urinary lead increased, ALA increased, and coproporphyrin decreased. The edetate test may be useful in assessing lead absorption in industrially and nonindustrially exposed individuals, and in the determination of lead deposition in the body.(26,33)

Therapy

Chelation of lead by the calcium salt of ethylenediaminetetraacetic acid, by calcium disodium edetate, or by penicillinamine is now the treatment of choice for inorganic lead and tetraethyllead intoxication. The chelated lead is excreted in the urine. It reduces lead body burdens in both soft and hard tissues. Edetate in doses of 0.5 to 2.5 g daily for an adult is given 6 to 12 hours intravenously. This dose may be repeated for 2 to 3 days with a 5 to 10-day rest period between courses. Nepropathy may result from the administration of larger doses than those mentioned above.

Penicillamine not only chelates lead but also supplies sulfhydryl groups. It appears to be as effective as oral edetate, can be taken orally, but can cause sensitization. A dose of 30 mg/kg up to a total of 2.0 g/day in four divided doses is satisfactory. Side effects include reduction of reticular iron, nephrotic syndrome, and agranulocytosis.(26,32-34)

ECONOMIC CONSIDERATIONS

As discussed in Volume I the comprehensive R&D planning and priority setting method requires the development of a quantitative measure of the impact of a stressor upon man in various categories of solution criteria. An attempt has been made under the lead model case study to obtain data on the health impact of the lead stressor and to reduce these data to dollar cost for possible direct comparison with impact in other selection criteria. Owing to its importance among the lead problems areas and the current national interest, the economic efforts were concentrated on the health impacts of pica.

Costs associated with lead poisoning can be placed into direct and indirect categories. Direct costs for both individuals and society would include those involved with detection, diagnosis, treatment, rehabilitation, custodial care, prevention, research, training, capital investment in medical facilities, and insurance. Expenditures for goods and services would be expected for physicians and other practitioners, hospitals, nursing homes, mental and other long-term-case facilities, drugs and therapy, medical laboratory testing, and supplies. Among the indirect costs of lead poisoning are wages and earnings lost during time off work, pension and relief payments, losses in taxes, and loss to the gross national product. When considering the wages lost, discounted life time earnings and cost of care should be recognized as well as the present values of future losses that constitute a portion of the total costs. Assembly of all of these types of costs was beyond the scope of the present program but costs were obtained for several of the categories to evaluate the validity of the approach and to provide some data for the estimation of urgencies in the lead case study.

Since acute cases of lead poisoning involve some hospital care, a range of estimates were developed for the total annual costs due to lead poisoning in the U. S. These estimates are summarized in Table 14. Lea Associates(43), who are involved in medical market research, estimate that 2000 patients per year are admitted to non-Federal general hospitals with primary diagnosis of lead poisoning (excludes outpatients, VA, and military hospitals), and that 19,000 patients are seen by M. D. 's in private practice. Another estimate of lead poisoning cases in short-term voluntary general hospitals is provided by the Professional Activity Study (PAS)(44). For nonmedical chemical substances, they report an incidence of 12 cases per 10,000 admissions with an average length of stay of 4 days. The American Hospital Association estimates that in 1969 there were 30 x 10^6 in-patient admissions to all U. S. general hospitals. If one-quarter of the admissions from nonmedical chemical substances were due to lead poisoning, the admissions per year would be 9,000; if one-sixth were due to lead, 6,000 admissions per year would be estimated. A third estimate of number of admissions due to lead poisoning is based upon Cook County Hospital data(45) and value from Goodman and Gilman(46). Cook County Hospital reported a total of 76,000 admissions in 1968 and Goodman and Gilman report that 100 to 150 admissions per year are due to lead poisoning. If an average of 25 admissions due to lead poisoning are assumed and the Cook County total admission data are extrapolated by direct proportion to the national admission figures, 50,000 cases of lead poisoning per year would be estimated. This estimate is believed to be too high by a factor of between 2 and 10 owing to the size of Cook County Hospital, the economic and social status of its patients, and to its location. The estimate reported in Table 14 was reduced accordingly. The expense figures used in Table 14 were derived from Table 15.

Another significant cost as a result of lead poisoning is that due to long-term care of individuals suffering brain damage. Urban ghetto children having pica are susceptible to lead poisoning from that route and a significant portion of those exposed suffer permanent brain damage. The cost of care and treatment of a brain damaged individual has been estimated to be \$200,000(47) or \$250,000(48). These are assumed to be lifetime costs. An estimate of annual incidence of brain damage from lead poisoning can be obtained as follows: approximately one-eighth of the U. S. population, 25 x 10^6 people, live in substandard housing in metropolitan, ghetto areas(49). It is then reasonable to assume that one-eighth of the children in the susceptible age group also live in ghetto areas. Lead poisoning is greatest in children between ages 1 and 3 years(36), and on the basis of population data there are 10 x 10^6 children in age group 1/2 to 3-1/2 years(50). Applying the one-eighth factor, there would be 1.25 x 10^6 children in risk areas. It has been reported that 10 to 25 percent of East Coast slum children have high blood-lead values(51). If 20 percent is a representative value, then it can be estimated that there can be 2.50 x 10^5 children with high blood-lead values in the U. S. Of the children with high blood-lead values, 2 to 5 percent develop symptomatic lead poisoning(51). Twenty-five percent of children with symptomatic lead poisoning suffer permanent brain damage(51), so the overall estimate for the U. S. would be 1.9 x 10^3 children suffering permanent brain damage.

An estimate can be made of the cost per year for treatment of children with brain damage from lead poisoning. Assuming that the 1.9 x 10^3 children represents an annual rate of incidence and that a steady-state exists between the rate of incidence and rate of death of affected individuals, then the annual costs would be:
$\$2.4 \times 10^5 \frac{\text{cost}}{\text{individual}} \times 1.9 \times 10^3 \frac{\text{children}}{\text{year}} = \$4.6 \times 10^8$ per year. This value is probably an upper limit in the sense that a steady-state may not exist. If the average life

TABLE 14. ESTIMATES OF HOSPITAL COSTS – LEAD POISONING

	Range		
	Low	Average	High
Number of Admissions/Year[a]	2000	8000	24000
Length of Stay – Number of Inpatient Days per Case (excludes long-term care)	2	5	10
Hospital Expenses per Patient/Day	\$30	\$45	\$60
Estimated Total Costs	$\$0.12 \times 10^6$	$\$1.8 \times 10^6$	$\$14.4 \times 10^6$

(a) See text: Lea, 2,000; PAS 3,000 to 9,000; Cook County Hospital projection 5,000 to 25,000.

TABLE 15. EXPENSE PER ADJUSTED PATIENT DAY FOR COMMUNITY GENERAL HOSPITAL, U. S.[a]

Year	Expense Patient Day, \$
1963	35.11
1964	37.58
1965	40.56
1966	43.66
1967	49.46
1968	55.80

(a) "Hospital Statistics", Journal of the American Hospital Association, Guide Issue, Part 2, 43(15), 469 (August, 1969).

expectancy of an afflicted individual is such that care and treatment are required over a 60-year period, then the cost per year per individual would be $\$2.4 \times 10^5/60 = \$4,000$.* The incremental costs per year for care and treatment of individuals suffering brain damage from lead poisoning is:

$\$4 \times 10^3 \dfrac{\text{cost}}{\text{individual}} \times 1.9 \times 10^3 \dfrac{\text{children}}{\text{year}} = \$7.6 \times 10^6$ per year. The current annual costs would range somewhere within these wide limits.

When considering lead poisoning resulting from pica, what are the costs to eliminate the source of the lead in the urban ghetto areas? The total costs are difficult to estimate for even such a simple approach since the possible sources must be located, entrance to the buildings must be gained, and the costs of the repainting must be borne. The latter costs are the easiest to estimate and give some order of magnitude to the total expected costs. It has been estimated(47) that the cost of removing paint from slum/ghetto dwellings could be $300 while replacement of doors and windowsills would range from $600 to 1200 per dwelling.

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* An assumed cost of $4,000 per year for care and treatment of an individual with brain damage appears reasonable. Available data(52) show that total expenses for long-term institutions for mental retardation in 1968 were $\$422.6 \times 10^6$ and that the average daily census was 118,316. The cost per year per patient based upon these data would be $3,572.

MATHEMATICAL MODELING AND RESULTS

Introduction

Although the environmental stressor lead is considered in detail in this model case study, it is pertinent to begin this section with a brief discussion of the generalized approach to mathematical model development, followed by the application of this approach to the stressor lead.

Mathematical models, in general, can be grouped into either statistical or deterministic models. Statistical models make use of statistical methods in order to determine the relationship between the dependent and independent variables and to determine the precision with which this relationship exists. In these methods, no attempt is made to identify a mechanism for the relationship and, because of the use of statistical methods, extensive experimental data are usually required.

An alternative procedure is to assume mechanisms for the relationships among the variables and to derive appropriate mathematical expressions for these mechanisms. By means of a few experiments on the system of interest or by study of a different system which is similar but has similar mechanisms, the values of the parameters in the relationships are determined. If the assumed relationships produce a model that adequately predicts the existing experimental data plus the data from verifying experiments, the model can be considered acceptable. Such models are called deterministic models. Deterministic models usuallyrequire much fewer data than the statistical models, however, follow-up experiments for validating the model are required in order to assure the adequacy of a deterministic model.

Deterministic models are usually derived in the form of systems of differential equations. The mathematical methods for solving differential equations can usually be grouped into three categories: analytical, semianalytical, or finite-difference methods. Analytical or closed-form solutions are limited to the cases where the equations are linear or the equations are of some very special form that can be reduced to linear equations by simple transformations. Also, there may be a practical limit on the size of the system of equations. Finite-difference methods have no theoretical limitations as to the type of equations or size of the system of equations which can be solved. However, the expense and time involved in solving equations requiring small differences can be limiting. Semianalytical solutions fall between analytical and finite-difference solutions in their relative merits. Their limitations as to size and linearity are slightly greater than finite-difference solutions, but they are much less limited than analytical solutions. Their chief advantage over finite-difference techniques is their increased speed of solution.

Generalized Mathematical Model Development

The development of mathematical models for EHS to utilize as management tools depends upon the detail and complexity of the systems that are being represented by the mathematical model. The hierarchical systems approach, which consists of defining the system of interest as a hierarchy in which the top echelon model covers all of the subject matter, is discussed in Volume I of this series of reports.

In the lower levels of the hierarchy of models in which lower echelon mathematical models are to be developed to represent the environmental stressor matrix, it is difficult to generalize and to develop a single mathematical model to encompass all stressors and which will predict the effect of these stressors upon man. Thus, models must be developed for each stressor or group of stressors. However, the integrated exposure to man from a stressor lends itself to generalization, and this integrated exposure can be represented by the following equation:

$$E_{ijk} = \int_{t_1}^{t_2} S_{ik}(t)\, P_{jk}(t)\, D_i(t)dt \quad , \tag{1}$$

where E_{ijk} is the exposure from stressor i over time t_1 to t_2 through pathway j to man at location k,

S_{ik} is the time varying source strength of stressor i at location k,

P_{jk} is the "pathway term" for pathway j and location k,

D_i is the exposure commitment rate for stressor i.

S_{ik} may be a smoothly varying function, a pulse, intermittent, a constant, etc., and is amenable to definition. P_{jk} is a fraction, less than one, representing the environmental dilution that a stressor may undergo. In the case of an external stressor such as noise, this dilution is simply a spatial spreading. With a material stressor, this dilution may be caused by many factors: water flow, meteorology, etc. Definition of the pathway terms is one of the major objectives in the development of the mathematical model. D_i is the exposure rate that man would be subjected to from one unit stressor intake from an ingestion standpoint or the presence of one unit of stressor from an ex ternal stressor (e.g., noise) and is amenable to definition.

Depending upon the detail desired to mathematically model the effect of a stressor upon man, each of the general terms discussed above, S_{ik}, P_{jk}, and D_i are amenable to representation by a mathematical model (submodel) which interacts with and becomes an input to the stressor model. For example, the source term, S_{ik}, can be modeled to yield a representation of emission of the stressor by the source considering processing that the stressor undergoes at the source, control devices that may be used, rates of release of stressor at the source, etc; the pathway term, P_{jk}, can be modeled to yield a representation of the environmental transport of the stressor from source to man by considering meteorological transport processes, ground and surface water processes, soil interactions, etc; and the exposure commitment term, D_i, can be modeled to yield a representation of the stressor's intake to deposition in and elimination from man by considering modes of entry of the stressor to man, assimilation into various organs, rates of elimination, etc.

At the preliminary stage of the mathe matical model, to mathematically represent the mechanisms involved that describe the relationships of the sources, S_{ik}, pathways, P_{jk}, and exposure commitment rates, D_i, for the stressor, ordinary differential equations are utilized because the primary interest is in demonstrating the framework of the model. Many phenomena cannot be accurately portrayed by ordinary differential equations, but the present state of the art does not justify oversimplification.

Pollution Chain Diagram

In order to represent the physical, chemical, and biological processes that operate in the real world, and which are elements of the terms, S_{ik}, P_{jk}, and D_i, as a mathematical representation of these processes, the stressor-environment-man relationship has to be determined. These relationships are derived fron the detailed pollution chain diagrams which are merely the qualitative representation of the pathways of stressor to man. Pollution chain diagrams can be formulated for each stressor or group of stressors, by utilizing existing data contained in the information data base. The type of data required may be completely qualitative, but should indicate the relationships of the source of the stressor with the environmental transport pathways for the stressor and the intake of the stressor into an individual.

An example of a pollution chain diagram for the environmental stressor lead is shown in Figure 4. This pollution chain diagram for the stressor lead was initially developed to present the environmental flow of lead from the sources to the receptor to guide the preliminary development of the mathematical model for lead so that the effect of the stressor lead could be predicted. Since the pollution chain diagram is a qualitative representation of the pathways of the stressor lead to man, the diagram was fairly easy to formulate. The qualitative information concerning lead that was contained in the information data base was used to formulate Figure 4. Even without very much qualitative information available in the information data base concerning a particular stressor, a pollution chain diagram can be developed by comparing that stressor with others of a similar nature, e.g., lead compared with other heavy metals.

Exercising the General Model

Development of the model is an iterative process of exercising the model in its preliminary development stages, determining at this preliminary stage what changes, if any, are necessary in the model in order to simulate the real world mechanisms, returning to the pollution chain diagram to investigate mechanisms that better represent the system, and querying the technical information base for generation of additional data that is required. When the model is utilized to predict the integrated exposure to man due to a stressor, the contribution of the stressor from each environmental pathway to an individual can be determined. Some pathways will be found to contribute significantly to the integrated exposure while some will contribute very little. The significant pathways should be investigated in more detail by obtaining better data from the technical information base and utilizing this data in the model while the insignificant pathways can be ignored at this stage in the model. Again this is an iterative process which becomes a closed loop between exercising the model, then based upon results obtained returning to the pollution chain diagram and technical data base to revise mechanisms, in order to represent better real world interactions mathematically and to obtain better data for pathways in the model that are significant contributors to output of the model. The iteration process may also include variations in the inputs to the model, such as source term as well as a sensitivity analysis of the parameters of the model to determine how sensitive the output of the model is to variations in the parameters of the model.

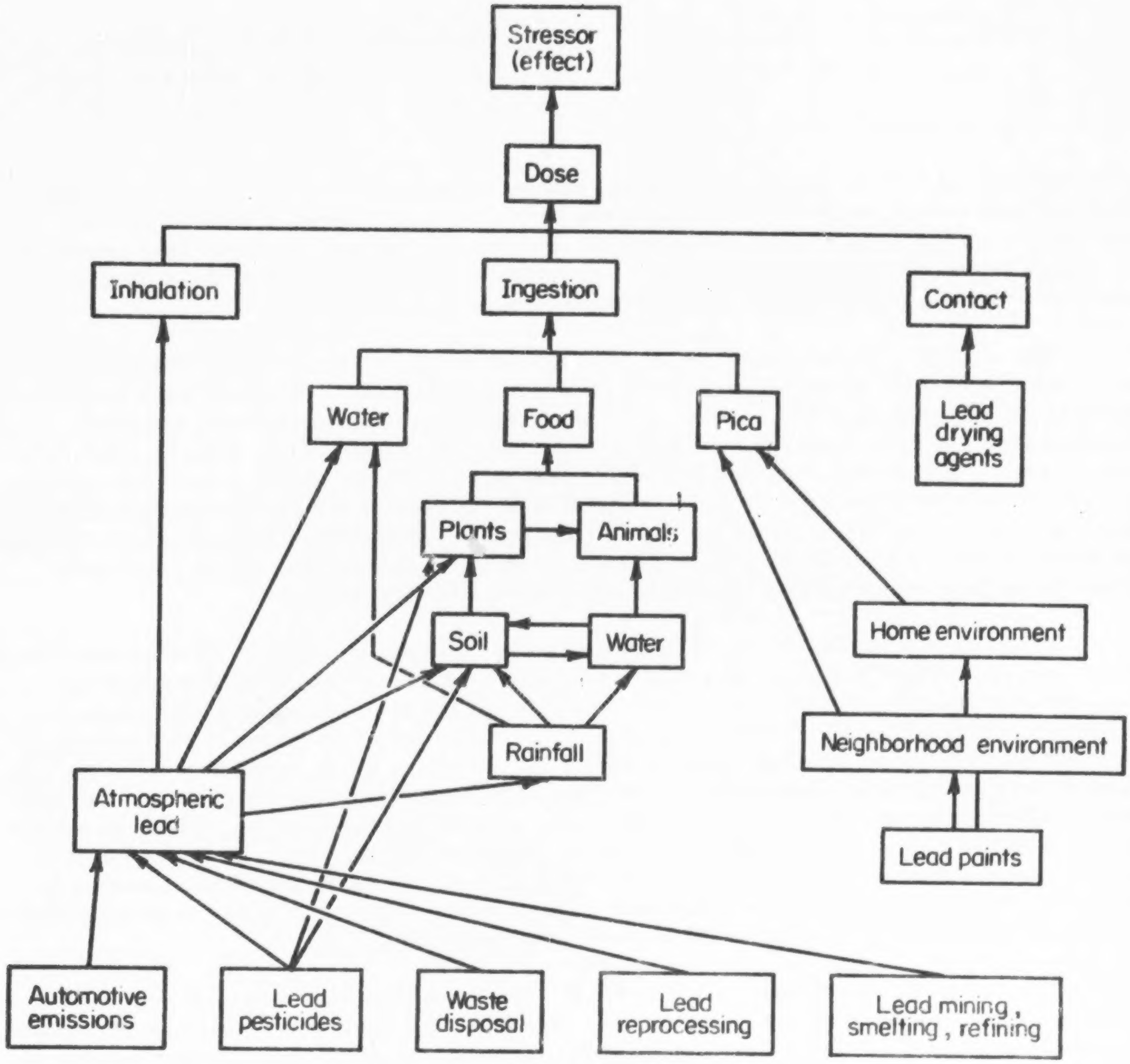
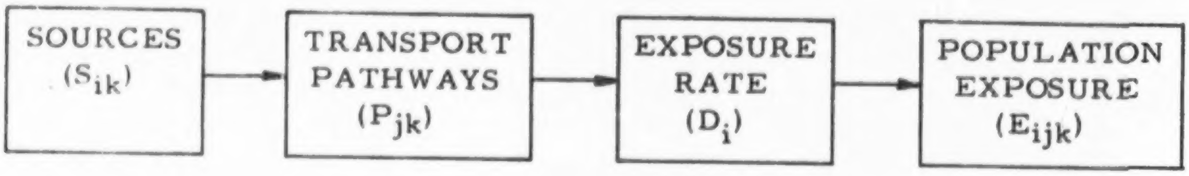

Stressor (effect)
Dose
Inhalation
Ingestion
Contact
Water
Food
Pica
Lead drying agents
Plants
Animals
Soil
Water
Home environment
Rainfall
Neighborhood environment
Atmospheric lead
Lead paints
Automotive emissions
Lead pesticides
Waste disposal
Lead reprocessing
Lead mining, smelting, refining

FIGURE 4. POLLUTION CHAIN DIAGRAM FOR THE ENVIRONMENTAL STRESSOR LEAD

Generalized Mathematical Model Applied to Lead

The generalized mathematical model expressed in Equation (1) can also be represented schematically as shown in the following block diagram:



The methodology of the generalized model can be extended to many specific stressors. For the lead model case study each of these terms is investigated to determine the mathematical and physical relationships between each term and the input data required to supply the basic information needs in order to develop the pollution chain diagrams and to develop and exercise the mathematical model.

The source term includes the amount of lead which is released into the environment and could ultimately become a stressor to the population. To determine the source term for lead, data related to the specific sources being investigated are required. Several types of data required for specific lead sources are listed as follows: the location of lead production plants and amount of lead produced; the location and lead usage of industries which use lead in their production processes or which incorporate lead into their products; the location and lead usage by the consumer of lead products such as in automobiles. Release rates of lead from these various sources can be calculated when these data are available from the information network analysis.

The transport pathways term includes the various environmental dilution processes which usually tend to decrease the concentration of a stressor such as lead from the time when the lead is released from the source until the lead becomes an exposure stressor to a population. This term can include few or many pathways. For example, the lead source term could be released to the environment as an airborne concentration, diluted with air, meteorologically transported, and then either deposited on ground surfaces, agricultural products, etc., or directly inhaled by a population. The lead source term could also be released with liquid waste, transported by water, and then be used as irrigation water or drinking water. Definition of these transport pathway terms is necessary to determine the environmental pathways and dilution in order to predict the exposure rates to a population from source term releases.

The exposure rate term for a stressor includes the various modes of exposure to the particular stressor such as, inhalation, ingestion, or absorption which are internal modes of exposure and the possibility of external exposure from the stressor outside of a body. For lead the significant modes of exposure are internal modes and include inhalation, ingestion, and absorption. The exposure rate term is the term which includes various parameters such as breathing rate, ingestion rate, body weight, organ weight, etc. This term is used to determine a population exposure due to any mode of exposure to the stressor and will be defined differently for various modes of exposure and various stressors.

The population exposure term is determined by integrating the body burden of the stressor over the time period of interest. For the stressor lead, the integrated exposure determined in this manner is not meaningful (μg Pb-days) since at the present time the effect on individuals from lead is estimated by correlating lead body burdens or lead organ concentrations with clinical medical effects. Therefore, the population exposure from lead which can be predicted using the mathematical model will be expressed as body organ concentrations.

The transport of the stressor lead through the ecosystem is simulated as a deterministic model and the resulting systems of differential equations are solved by analytical techniques. The solution of the systems of differential equations which comprise the mathematical model for the environmental stressor lead yields the determination of the dependent variables (e. g. , concentration or quantity of stressors in air, water, body tissue, etc.) with respect to the independent variables (e. g. , time and distances from some reference point). The parameters in the differential equations, such as, various rate coefficients, velocities, dietary intake rates, constants, etc. , are obtained (1) from data stored in the technical information base, (2) by calculation from data stored in the technical information base, or (3) by extrapolation or estimation from data stored in the technical information base when the necessary data is unavailable. Thus, the formulation of the mathematical model which simulates transport of the stressor lead through the ecosystem and which predicts the lead concentrations in air, water, body tissue, etc. , indicates the specific data which must be acquired. For example, the "normal" concentrations and the assimilation rate coefficients of the stressor lead in all body organs of interest are data that are required in order to predict the response of an individual (changes in lead organ concentrations) to varying intakes of lead via ingestion or inhalation. As stated, some of these parameters in the differential equations are available in the information base. When the data are not available, then the data can be calculated, estimated, or extrapolated.

The input data for the model should always be as accurate as possible. However, when insufficient data exist to formulate the mathematical model and estimates of data are made, these estimates may be accurate to only a factor of two or possibly even an order of magnitude, but are always estimated conservatively (so that predicted values are always greater than real values).

It should be recognized that the degree of confidence in the results of the simulation developed from limited data used in the preliminary mathematical model may not be high. Such a simulation though can be used to identify the major factors affecting the response of an individual to intake of lead and, in this way, guide the data acquisition toward those areas needing further study (e. g. , airborne concentrations of lead). As the data on those areas needing further study become available, they can be used in a more comprehensive model, and the degree of confidence will increase. Furthermore, when specific pathways are determined to be the major contributor to the predicted levels of lead in body organs, these pathways are then investigated in more detail to obtain better data for these pathways.

The approach used to develop the mathematical model for the lead case study was to represent the transport of lead from the source to an individual with the subsequent distribution of lead in several body organs of the individual due to the two principal modes of entry into the body, inhalation and ingestion. Since the ingestion of lead by foodstuffs does not vary significantly by location or by types of foodstuff, at this preliminary stage of mathematical model development for the stressor lead, only the atmospheric lead transport model was developed.

Lead in the Body Model

The schematic representation of the model (submodel for lead in the body) is shown in Figure 5. This lead in the body model is the part of the mathematical model that describes the distribution of lead in several body organs (lung, blood, total body, bone, liver, and kidneys due to intake of lead by an individual from inhalation and ingestion and is based on the concept of a "Standard Man" as used by ICRP$^{(53)}$. The following differential equations represent the time varying quantities of lead in these several body organs:

$$\dot{Y}_L = DBC_{AI} - \lambda_L Y_L \tag{2}$$

$$\dot{Y}_{BL} = (f_1 E+G)\,BC_{AS} + f_1 FBC_{AI} + f_1 \sum_{n=1}^{N} I_n C_n + \lambda_L Y_L - \lambda_{BL} Y_{BL} \tag{3}$$

$$\dot{Y}_{TB} = (f_w)_{TB}\left[\sum_{n=1}^{N} I_n C_n + FBC_{AI}\right] + (f_a)_{TB}\,BC_{AS} + (f_2)_{TB}\lambda_L Y_L - \lambda_{TB} Y_{TB} \tag{4}$$

$$\dot{Y}_B = (f_w)_B\left[\sum_{n=1}^{N} I_n C_n + FBC_{AI}\right] + (f_a)_B BC_{AS} + (f_2)_B \lambda_L Y_L - \lambda_B Y_B \tag{5}$$

$$\dot{Y}_{LI} = (f_w)_{LI}\left[\sum_{n=1}^{N} I_n C_n + FBC_{AI}\right] + (f_a)_{LI} BC_{AS} + (f_2)_{LI}\lambda_L Y_L - \lambda_{LI} Y_{LI} \tag{6}$$

$$\dot{Y}_K = (f_w)_K\left[\sum_{n=1}^{N} I_n C_n + FBC_{AI}\right] + (f_a)_K BC_{AS} + (f_2)_K \lambda_L Y_L - \lambda_K Y_K \tag{7}$$

where

$\dot{Y}_L$, $\dot{Y}_{BL}$, $\dot{Y}_{TB}$, $\dot{Y}_B$, $\dot{Y}_{LI}$, and $\dot{Y}_K$ are the derivatives with respect to time of the lead quantity in the lung, blood, total body, bone, liver, and kidney, respectively, μg/day

Y_L, Y_{BL}, Y_{TB}, Y_B, Y_{LI}, and Y_K are the quantity of lead at time t in the lung, blood, total body, bone, liver, and kidney, respectively, μg

C_A is the airborne lead concentration, where the subscript S is soluble lead compounds and the subscript I is insoluble lead compounds, μg/cm^3

I_n is the ingestion rate of foodstuff n, g/day

C_n is the concentration of lead in foodstuff n, μg/g

B is the breathing rate of Standard Man, cm^3/day

D is the fraction of insoluble lead retained in the lung (dimensionless)

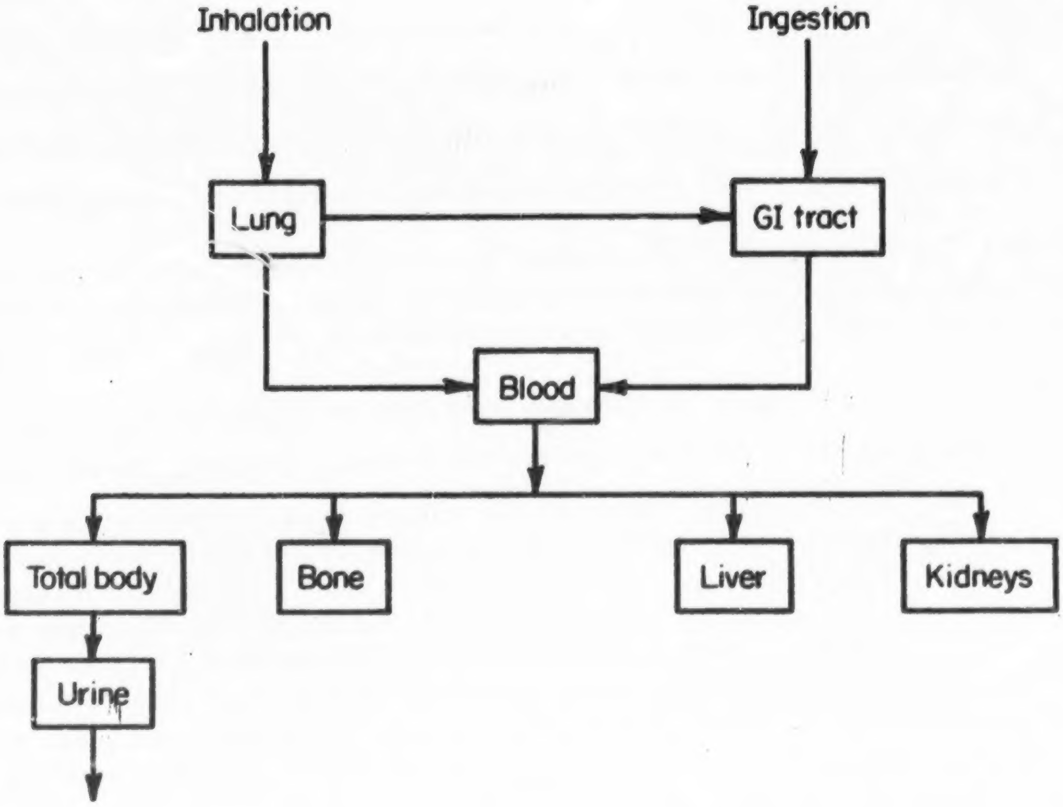

Inhalation
Ingestion
Lung
GI tract
Blood
Total body
Bone
Liver
Kidneys
Urine

FIGURE 5. LEAD IN BODY MODEL

F is the fraction of insoluble lead which is deposited in upper respiratory passages of the lung and subsequently swallowed (dimensionless)

E is the fraction of soluble lead which is deposited in upper respiratory passages of the lung and subsequently swallowed (dimensionless)

f_1 is the fraction of lead ingested which reaches the blood (dimensionless)

λ_L, λ_{BL}, λ_{TB}, λ_B, λ_{LI}, and λ_K are the elimination rate coefficients for lead from the lung, blood, total body, bone, liver, and kidney, respectively, days^{-1}

$(f_2)_{TB}$, $(f_2)_B$, $(f_2)_{LI}$, and $(f_2)_K$ are the fractions of lead transferred from blood to total body, bone, liver, and kidney, respectively (dimensionless)

$(f_a)_{TB}$, $(f_a)_B$, $(f_a)_{LI}$, and $(f_a)_K$ are the fractions of lead inhaled reaching the total body, bone, liver, and kidney, respectively (dimensionless)

$(f_w)_{TB}$, $(f_w)_B$, $(f_w)_{LI}$, and $(f_w)_K$ are the fractions of lead ingested reaching the total body, bone, liver, and kidney, respectively (dimensionless).

Assuming that the lead concentration in various organs of man are in equilibrium with lead intake rates due to inhalation of ambient airborne lead concentrations and ingestion of average lead concentrations in foodstuffs ($\dot{Y}_L = \dot{Y}_{BL} = \dot{Y}_{TB} = \dot{Y}_B = \dot{Y}_{LI} = Y_K = 0$), Equations (2) through (7) may be rewritten to solve for Y_L, Y_{BL}, Y_{TB}, Y_B, Y_{LI} and Y_K, the quantity of lead at equilibrium time in the lung, blood, total body, bone, liver, and kidney, respectively. Most of the values used for the various parameters, constants and elimination rate coefficients for these calculations were selected from the ICRP Report(53). The values for C_A were found in the U. S. Public Health Service three cities study(32) and in studies by Kehoe(54). The values for $\Sigma I_n C_n$ were reported by Kehoe(55). The elimination rate coefficient for lead from blood was calculated by fitting to straight line curves, by the least squares method, reported experimental data for lead concentrations in blood as a function of lead inhalation and lead ingestion. The steady-state form of Equation (3), i. e., $Y_{BL} = 0$, indicates a linear relation between Y_{BL} and C_A, over the range of these measurements for C_A.

Measurement of lead concentrations in the blood is one clinical method of detecting intake of lead by either inhalation or ingestion. Assuming that the lead concentrations in various organs of man are in equilibrium with lead intake rates due to inhalation of ambient airborne lead concentrations and ingestion of average lead concentrations in foodstuffs and using several reported average airborne lead concentrations and an average lead intake due to ingestion, the lead levels in several organs of man were calculated. The blood-lead levels using this theoretical model were in the range of the experimentally measured blood-lead levels and the values determined by the empirical correlations. This agreement, as shown in Table 16, provides a modest degree of validation of the model. Thus, the model can be used with some confidence to predict lead levels in other organs and to predict the transient response of these organs to changing environmental lead levels. However, the blood lead content calculated in this model is assumed to be linear with respect to blood-lead assimilation rates, due to lack of data supporting a nonlinear assimilation rate of lead into the blood from intake via ingestion and inhalation. Thus, when airborne lead concentrations are higher than average (i. e., approximately greater than 10 μg/m^3 or the amount of lead ingested daily is greater than several mg lead per day (the pica case), the calculated blood lead content will be conservatively high.

TABLE 16. MEASURED AND PREDICTED BLOOD-LEAD LEVELS

Type of Population	Estimated Average Respiratory Exposure, μg/m^3	Mean Blood-Lead Level[a], μg/100 g	Predicted Blood-Lead Level[b], μg/100 g
Composite Rural U. S.	0.5	16	16.5
Composite Urban U. S.	1.0	21	17.6

(a) U. S. Public Health Service, "Survey of Lead in the Atmosphere of Three Urban Communities", Publ. No. 999-AP-12 (1965).

(b) Predicted from lead model and includes average daily ingestion of 0.3 mg lead.

In addition to calculating blood-lead levels, due to inhalation and ingestion of lead at equilibrium conditions, lead levels can be calculated in the blood and other organs for time varying intakes of lead via inhalation and ingestion. The time varying quantities of lead in the blood can be calculated as follows:

$$Y_{BL} = \frac{K}{\lambda_{BL}}\left[1 - e^{-\lambda_{BL}t}\right] + b_1 e^{-\lambda_{BL}t} + DBC_{AI}\left[\frac{1}{\lambda_{BL}} + \frac{e^{-\lambda_L t}}{\lambda_L - \lambda_{BL}} - \frac{\lambda_L e^{-\lambda_{BL}t}}{\lambda_{BL}(\lambda_L - \lambda_{BL})}\right] + \frac{a_1\lambda_L}{\lambda_L - \lambda_{BL}}\left[e^{-\lambda_{BL}t} - e^{-\lambda_L t}\right] , \qquad (8)$$

where

$$K = f_1 EBC_{AS} + f_1 FBC_{AI} + GBC_{AS} + f_1 \sum_{n=1}^{N} I_n C_n$$

a_1 is the quantity of lead at the initial time t = 0 in the lung, μg

b_1 is the quantity of lead at initial time t = 0 in the blood, μg.

Both the time varying blood lead levels as calculated by Equation (8) and the equilibrium time blood level as calculated by Equation (3) require as input values the airborne concentrations of lead by inhalation (C_A) and the quantity of lead ingested ($\Sigma I_n C_n$). The average daily intake of lead by ingestion of foodstuffs, though slightly variable, is assumed to be 300 micrograms of lead per day. This intake of lead by foodstuffs does not vary significantly by location or by types of foods being eaten. However, the average daily intake of lead by inhalation is due to airborne lead concentrations which may be variable depending upon geographical area in the country. Airborne lead concentrations are assumed to be due mainly to automobile exhausts of lead.

Atmospheric Lead Transport Model

The airborne concentrations of lead that might be inhaled by an individual, the "recepter", due to the release and steady state airborne transport of lead from several types of sources can be calculated using several equations. The model (submodel) to represent the steady state airborne transport of lead investigated the following sources: (1) a continuous point source emission (a stack), (2) a crosswind line source emission (a highway), (3) a down wind line source emission (a highway), and (4) an area source emission (a city).

The development of these meteorological transport model equations was based upon Sutton's equations for a continuous point source(56) since the transport of lead over long distances (how one city's lead emission affects the airborne lead concentrations of another city) was desired. The line and area source equations were developed by superpositioning an indefinite number of sources along the line or area respectively and integrating these sources over the limits of these regions. The integrations for the cross wind line source equation was exact, whereas, the integration for the downwind line source and area source equations were approximate integrations. The equations for meteorological transport for the four sources are listed below:

Continuous point source

$$\chi = \frac{2Q}{\pi C^2 u x^{2-n}} \exp \left[- \frac{y^2 + h^2}{C^2 x^{2-n}} \right] \qquad (9)$$

Finite crosswind line source

$$\chi = \frac{Q_L \exp \left[- \frac{h^2}{C^2 x^{2-n}} \right]}{C u x^{1-n/2} (\pi)^{1/2}} \left[\text{erf} \left[\frac{H-y}{C x^{1-n/2}} \right] + \text{erf} \left[\frac{H+y}{C x^{1-n/2}} \right] \right] \qquad (10)$$

Finite downwind line source

$$\chi = \frac{2Q_L}{\pi C^2 u (2-n)} \left[\frac{y^2 + h^2}{C^2} \right]^{\frac{n-1}{2-n}} \left[\Gamma \left(\frac{1-n}{2-n} \right) \right] \text{ for } 0 < x < L \qquad (11a)$$

$$\chi = \frac{2Q_L x^{n-1}}{\pi C^2 u} \left[\frac{L}{x-L} \right] \exp \left[- \frac{h^2 + y^2}{C^2 x^{2-n}} \right] \text{ for } x > L \qquad (11b)$$

Finite areal source

$$\chi = \frac{4HQ_R}{(2-n)\pi C^2 u} \left[\frac{(y-H)^2 + h^2)}{C^2} \right]^{\frac{n-1}{2-n}} \left[\Gamma \left(\frac{1-n}{2-n} \right) \right] \text{ for } 0 < x < L \qquad (12a)$$

$$\chi = \frac{4Q_R\, x^{n-1} HL}{\pi\, C^2 u(x-L)} \; \exp\left[-\frac{h^2 + (y-H)^2}{C^2\, x^{2-n}}\right] \text{ for } x > L \;, \qquad (12b)$$

where

χ	is the airborne lead concentration at point x downwind from the source, g/m^3
Q, Q_L, Q_R	are the lead emission rates from the point source, the line sources, and the area source, respectively, g/day, g/day/m, $g/day/m^2$
x, y	are downwind and crosswind coordinates measured from a ground point beneath a continuous source, m
h	is the height of source above ground level, m
u	is the mean wind speed, m/day
C	is generalized diffusion coefficient, $(m)^{n-2}$
n	is the parameter associated with stability (dimensionless)
H	is the crosswind coordinate (half-width) measured from the center point of the crosswind finite line source and the areal source, m
L	is downwind coordinate (full length) measured from the beginning point of the downwind finite line source and the areal source, m
erf (n)	is the error function [listed in Handbook of Mathematical Functions, Ed. by M. Abramowitz and I. A. Stegun, NBS Applied Math Series, 55 (1964)]
Γ (n)	is the gamma function [listed in Handbook of Mathematical Functions Ed. by M. Abramowitz and I. A. Stegun, NBS Applied Math Series 55 (1964)].

Several mathematical diffusion models have been reported in the last few years for application in specific metropolitan areas(57, 58, 59). The resolution of each of these models is sophisticated to a degree which requires a large amount of data from the specific metropolitan region of interest and each of these models have been computerized to perform the many calculations included in each model. Both the Travelers Research Corporation model (TRC)(58) and the Argonne National Laboratory model (ANL)(59) are short time period models (daily predictions of concentrations). The Martin-Tikvart model (MT)(57) permits calculation of seasonal or monthly average concentrations of any air pollutant.

Since the resolution of the atmospheric transport model required for this study was low, the simple atmospheric transport model for lead as expressed in Equations (9) through (12) was developed and used The data required for Equations (9) through (12) were readily available or readily estimated, whereas, a large effort would have been required to obtain the necessary data for the TRC, ANL, or MT models and to adapt the computer programs for these models to the Battelle-Columbus computer.

Results

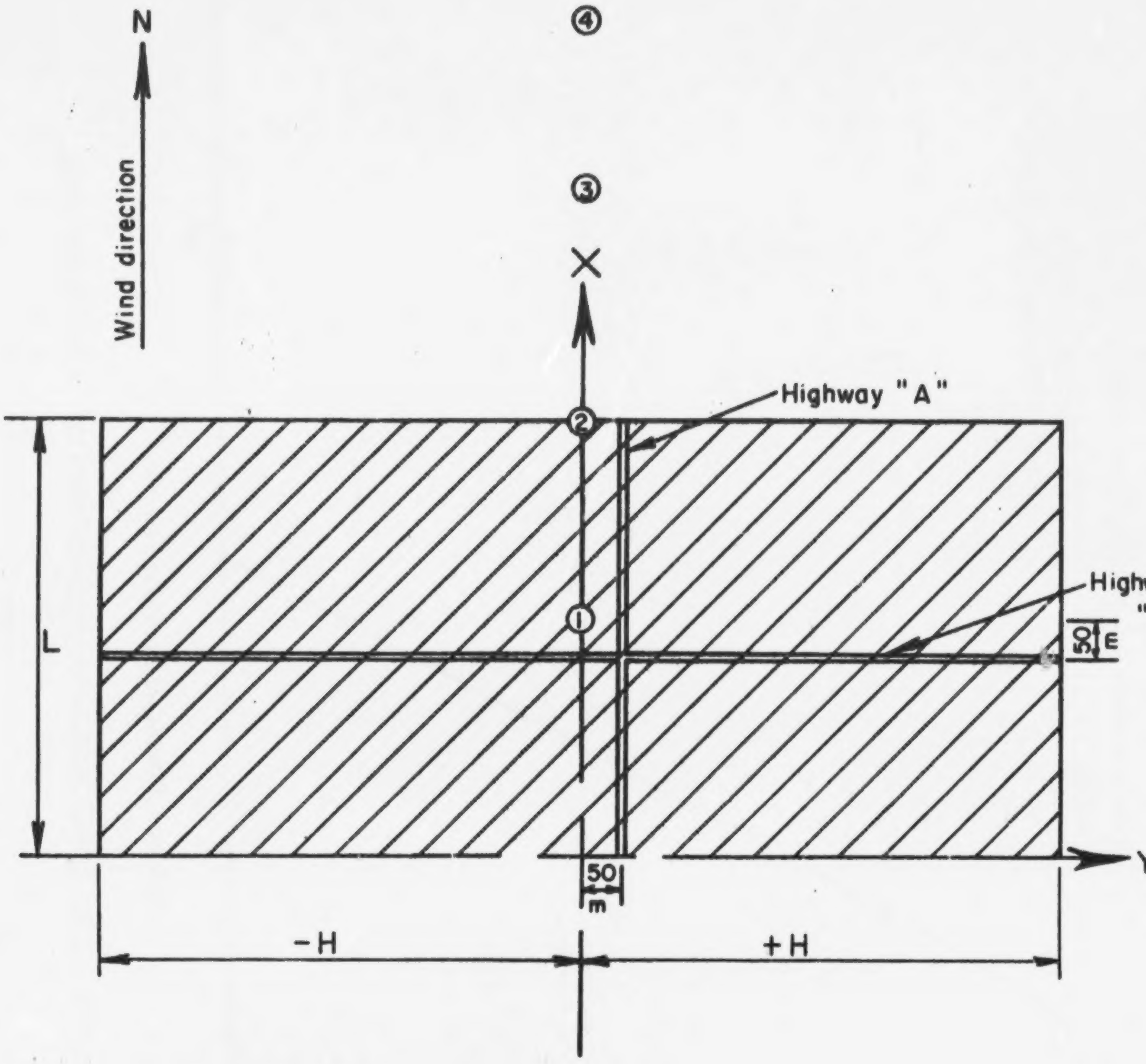
The mathematical model developed to represent the environmental transport of lead from source into man is a tool which provides a quantitative investigation of lead concentrations in real world systems, and of particular interest, airborne lead concentrations, foodstuff lead concentrations, and body organ lead concentrations. Since the inhalation and ingestion modes of intake to man are the two major ways in which lead is taken into the body, each of these modes of intake of lead were investigated by utilizing the mathematical model developed for the environmental stressor lead.

The inhalation mode of entry was examined by calculating the concentrations of a airborne lead using Equations (9) through (12) at various distances from four types of sources and at various heights of these sources above the ground surface: (1) a point source, (2) downwind line source, (3) crosswind line source, and (4) area source. A schematic drawing of these sources is shown in Figure 6. A unit emission rate from these sources of 1 g/day, 1 g/m/day, 1 g/m/day, and 1 g/m^2/day, respectively, was used to calculate the airborne lead concentrations due to these sources. Curves for airborne lead concentrations vs distance from the source for each of these sources are shown in Figures 7, 8, 9 and 10 for an average weather condition of turbulence, or mixing, and for a height of 0.25 m above ground surface (the approximate distance of an automobile exhaust above the ground surface). Values for the diffusion coefficients, C, were obtained from Eisenbud(60) while the mean wind speed data were obtained from USPHS Three Cities Study(32). The airborne lead concentrations from any source with similar emission and weather conditions can be calculated from the curves in Figures 7-10 by applying the ratio of actual source emission rate to the unit source emission rate used to generate these curves.

To investigate the airborne lead concentrations in a particular geographical locality, a Midwestern city, Cincinnati, Ohio, was chosen. It was assumed that the major contributor to atmospheric lead in Cincinnati is the exhausts of automobiles. Therefore, the line and area sources were used to calculate airborne lead concentrations due to: (1) automobiles randomly traversing city streets (assuming even distribution over the city) as an area source and (2) automobiles concentrated on a major freeway as a line source (both downwind and crosswind freeways). Curves of airborne lead concentrations vs distance from the source were generated for these three sources for Cincinnati and are shown in Figures 11, 12, and 13.

The emission rates of these sources were calculated as follows:

In the 1969 Market Guide(61) estimates of dollar volume of gasoline sales for 1969 in Cincinnati were $67.9 million. Assuming (a) 3 gallons of gasoline is purchased per dollar of sales, (b) 2.4 grams of lead is contained in each gallon of gasoline, (c) 33 percent of lead consumed in the automobile is exhausted to the atmosphere, and (d) 50 percent of exhausted lead becomes airborne; then approximately one-half million grams of lead per day from automobile exhausts becomes airborne. This quantity distributed over the area of Cincinnati, represents the area source emission rate. Since the wind direction is usually from the south in Cincinnati, the downwind line source emission rate is this quantity of lead released by automobiles each day distributed over the length of the major freeway travelling in a north-south direction while the crosswind line source emission rate is this quantity of lead released by automobiles each day distributed over the length of the major freeway travelling in an east-west direction.


N
Wind direction
④
③
X
Highway "A"
②
Highway
"B"
①
50 m
L
Y
50 m
−H
+H

Legend

Highway "A" ≡ Downwind line source
Highway "B" ≡ Crosswind line source
▨ ≡ Area source
① ≡ Locations of individuals for selected calculations of airborne lead concentrations

FIGURE 6. SCHEMATIC REPRESENTATION OF SEVERAL LEAD SOURCES

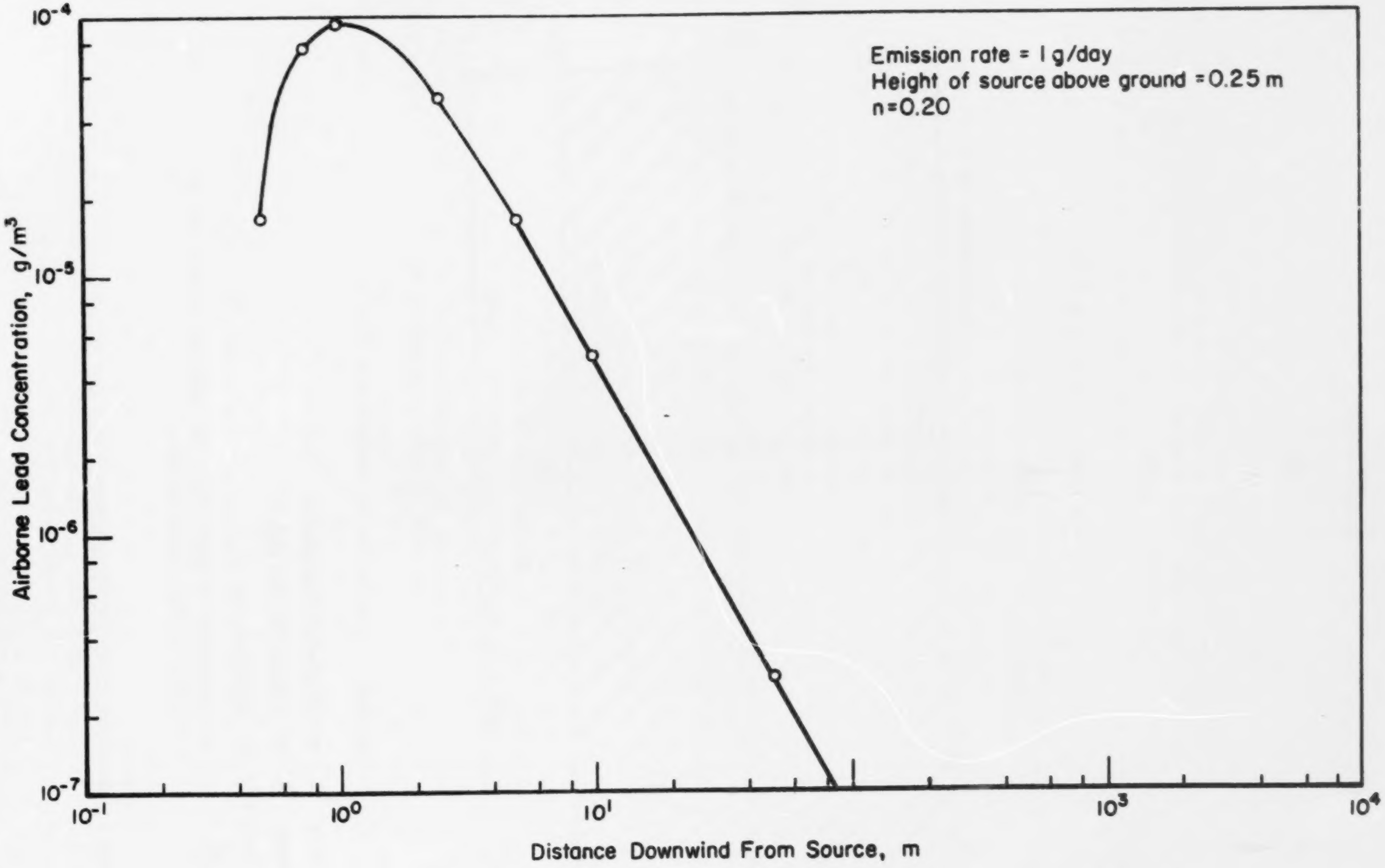


FIGURE 7. AIRBORNE LEAD CONCENTRATIONS DOWNWIND OF A POINT SOURCE

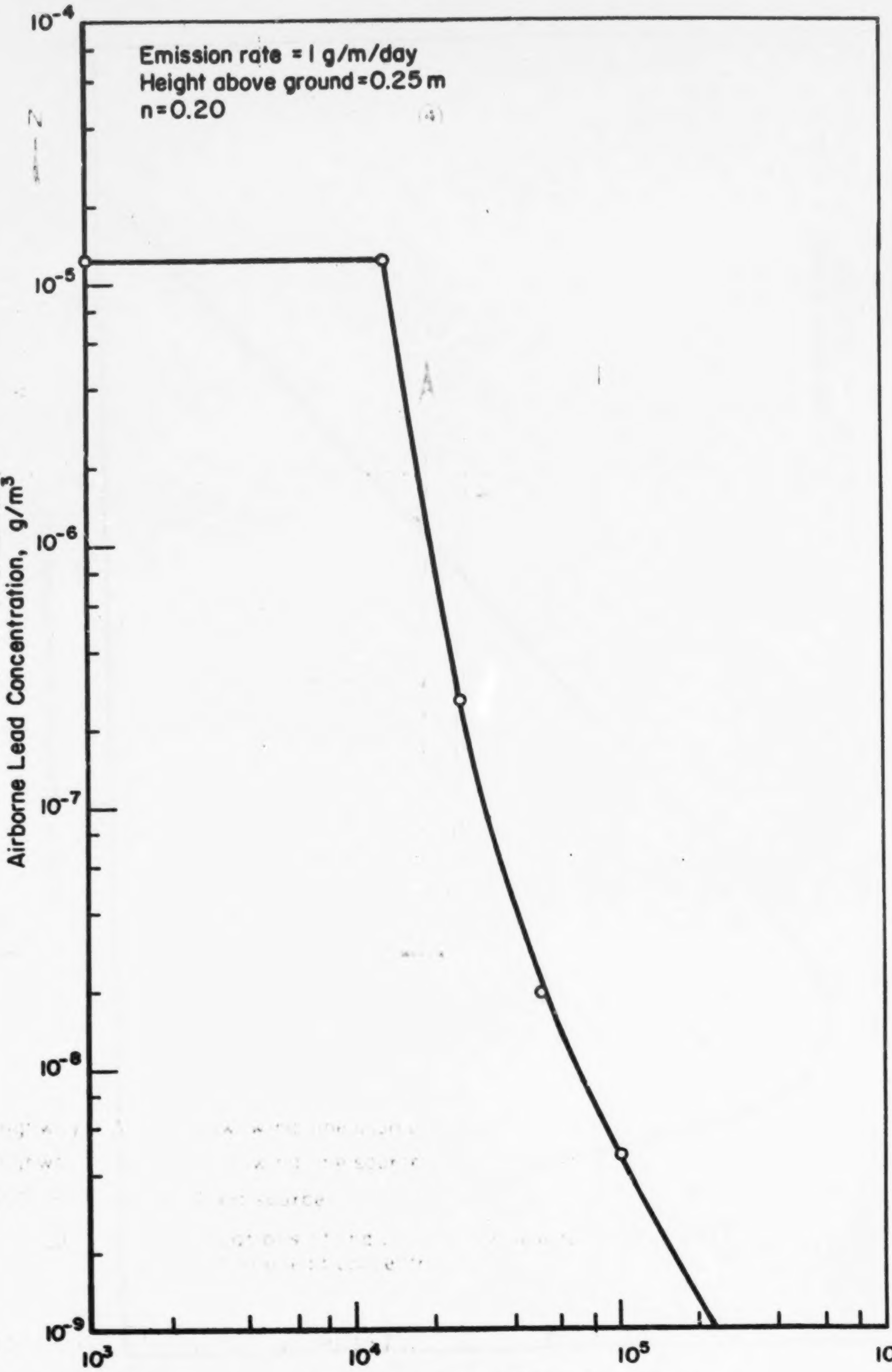


FIGURE 8. AIRBORNE LEAD CONCENTRATIONS DOWNWIND OF A DOWNWIND LINE SOURCE

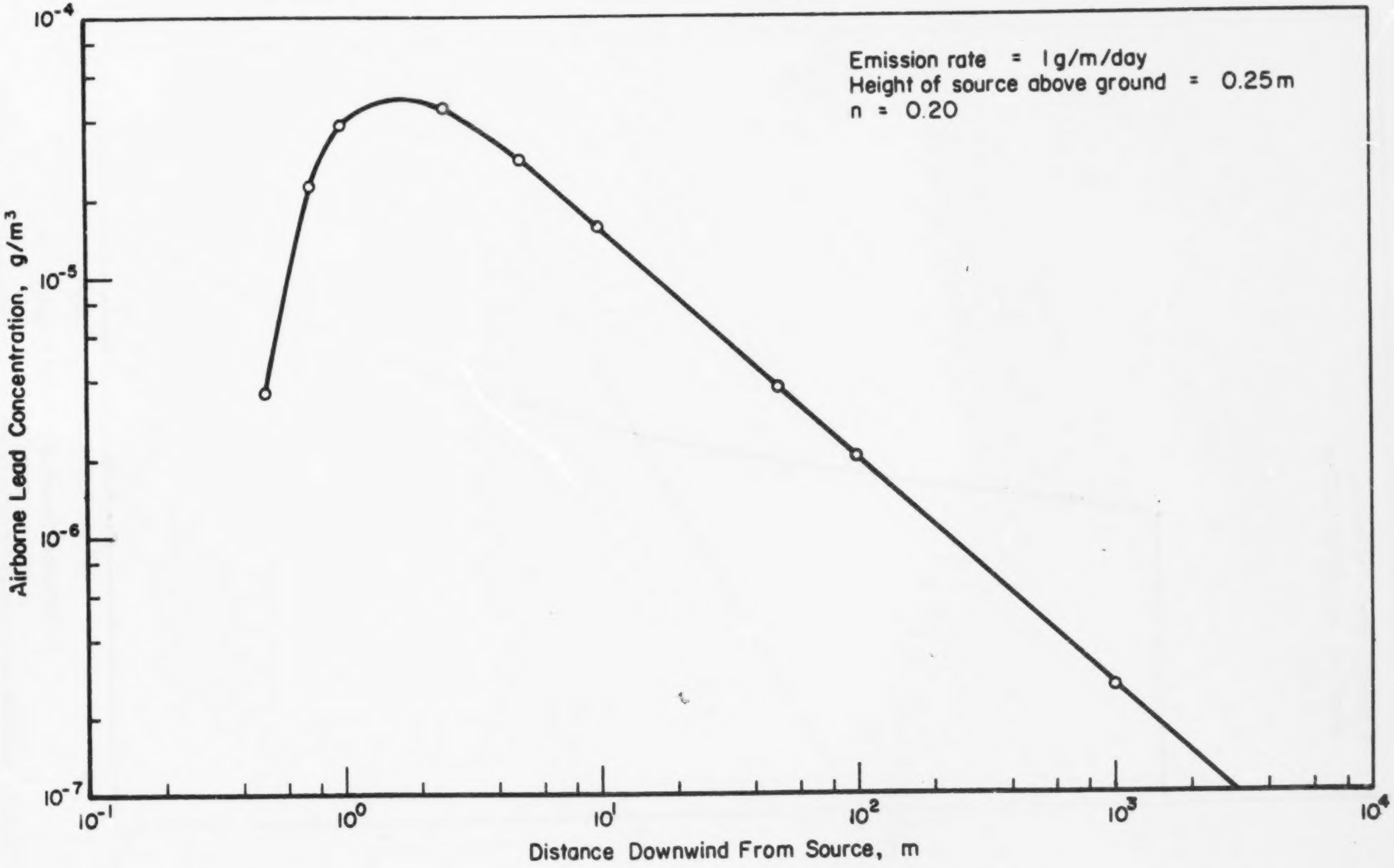


FIGURE 9. AIRBORNE LEAD CONCENTRATIONS DOWNWIND OF A CROSSWIND LINE SOURCE

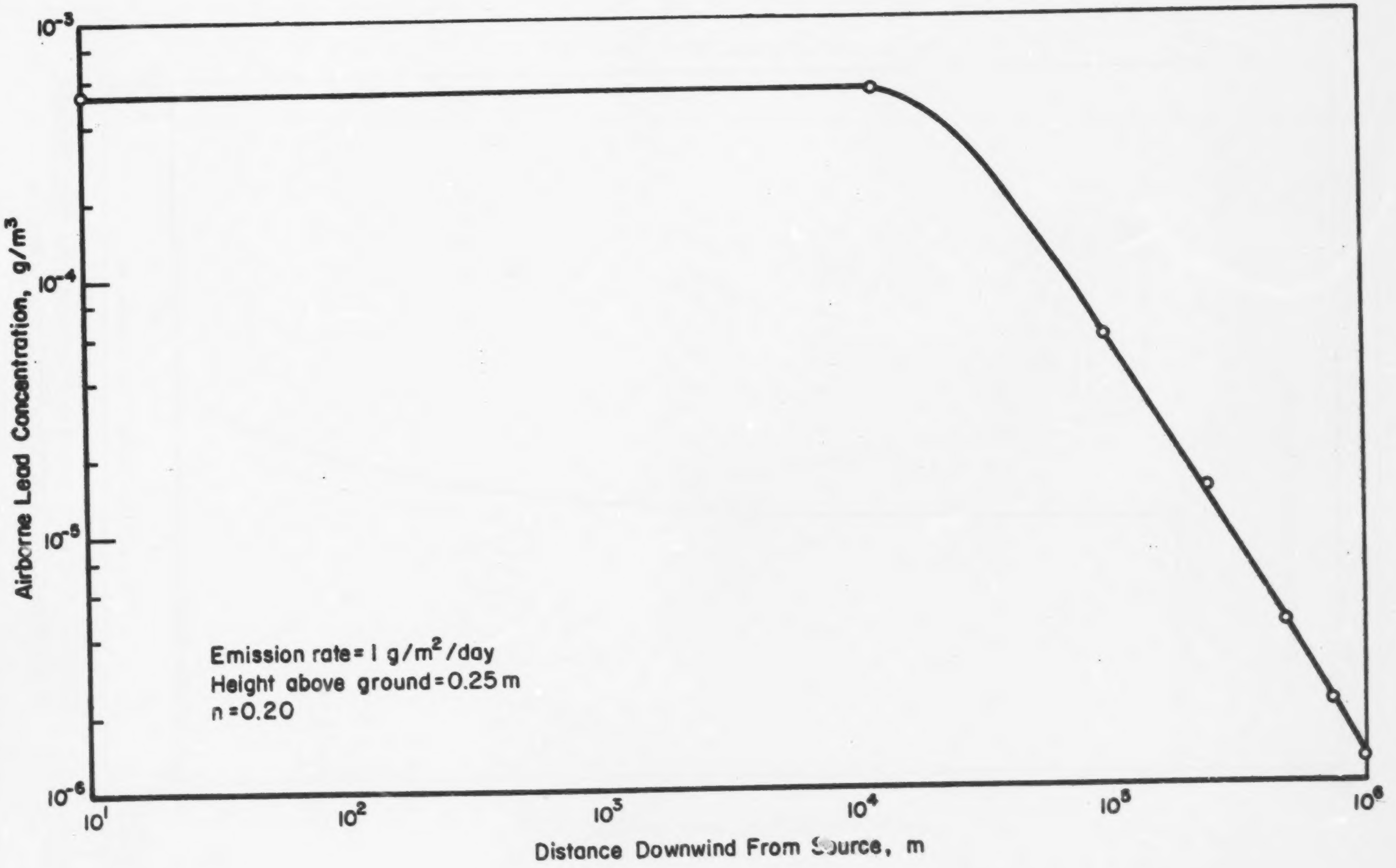


FIGURE 10. AIRBORNE LEAD CONCENTRATIONS DOWNWIND OF AN AREA SOURCE

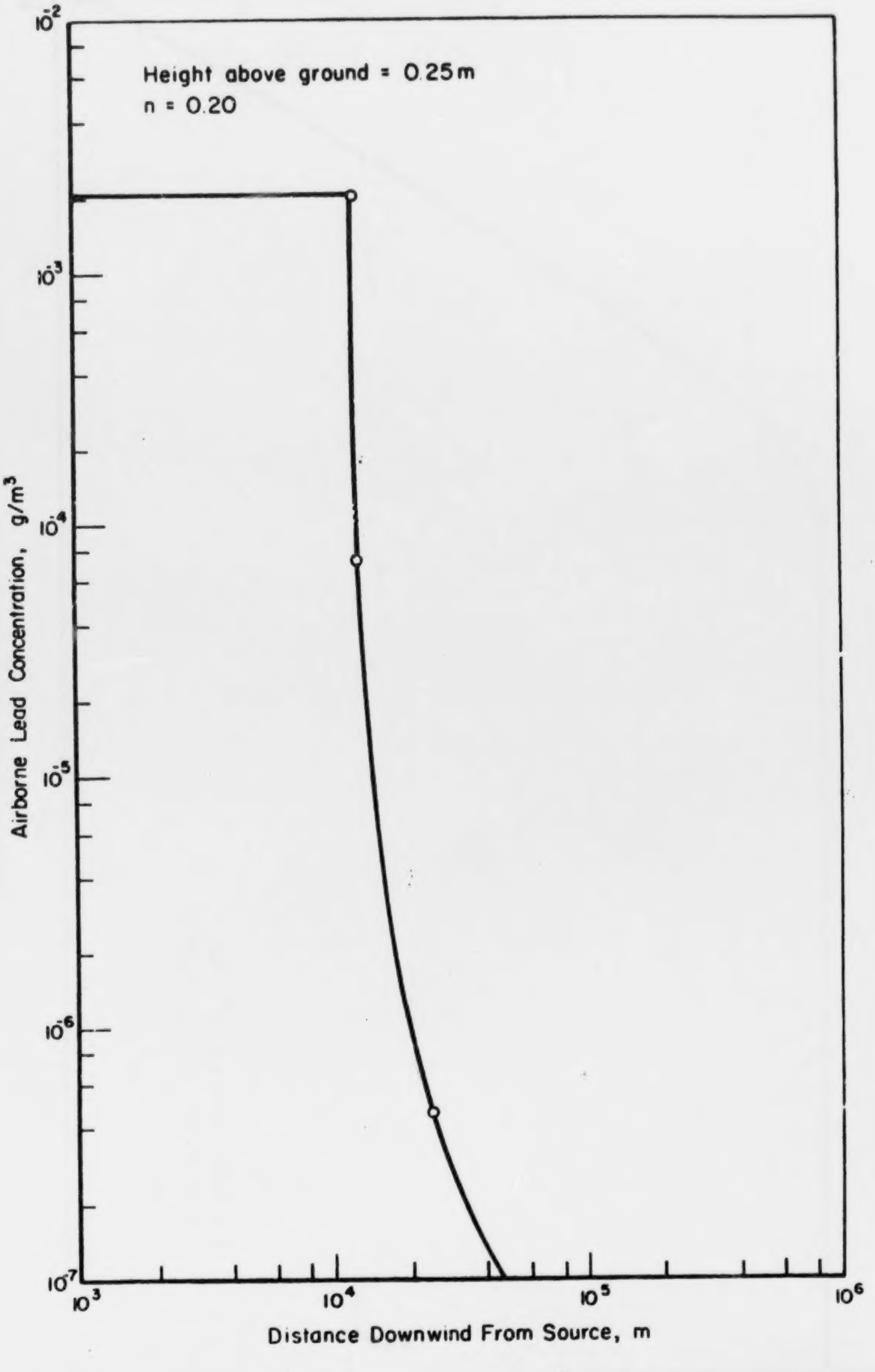


FIGURE 11. AIRBORNE LEAD CONCENTRATIONS IN CINCINNATI DOWNWIND OF A DOWNWIND LINE SOURCE

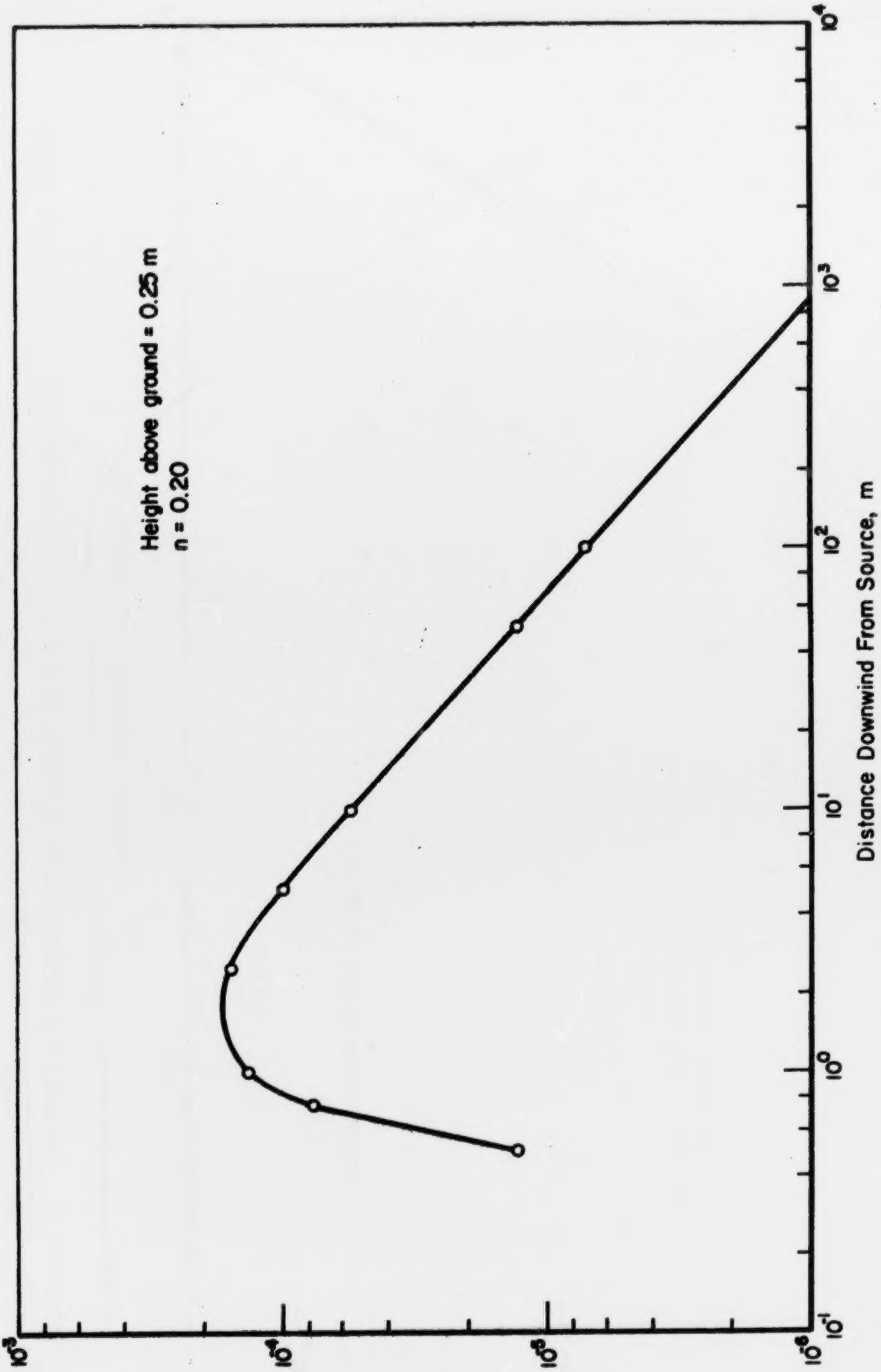


FIGURE 12. AIRBORNE LEAD CONCENTRATIONS IN CINCINNATI DOWNWIND OF A CROSSWIND LINE SOURCE

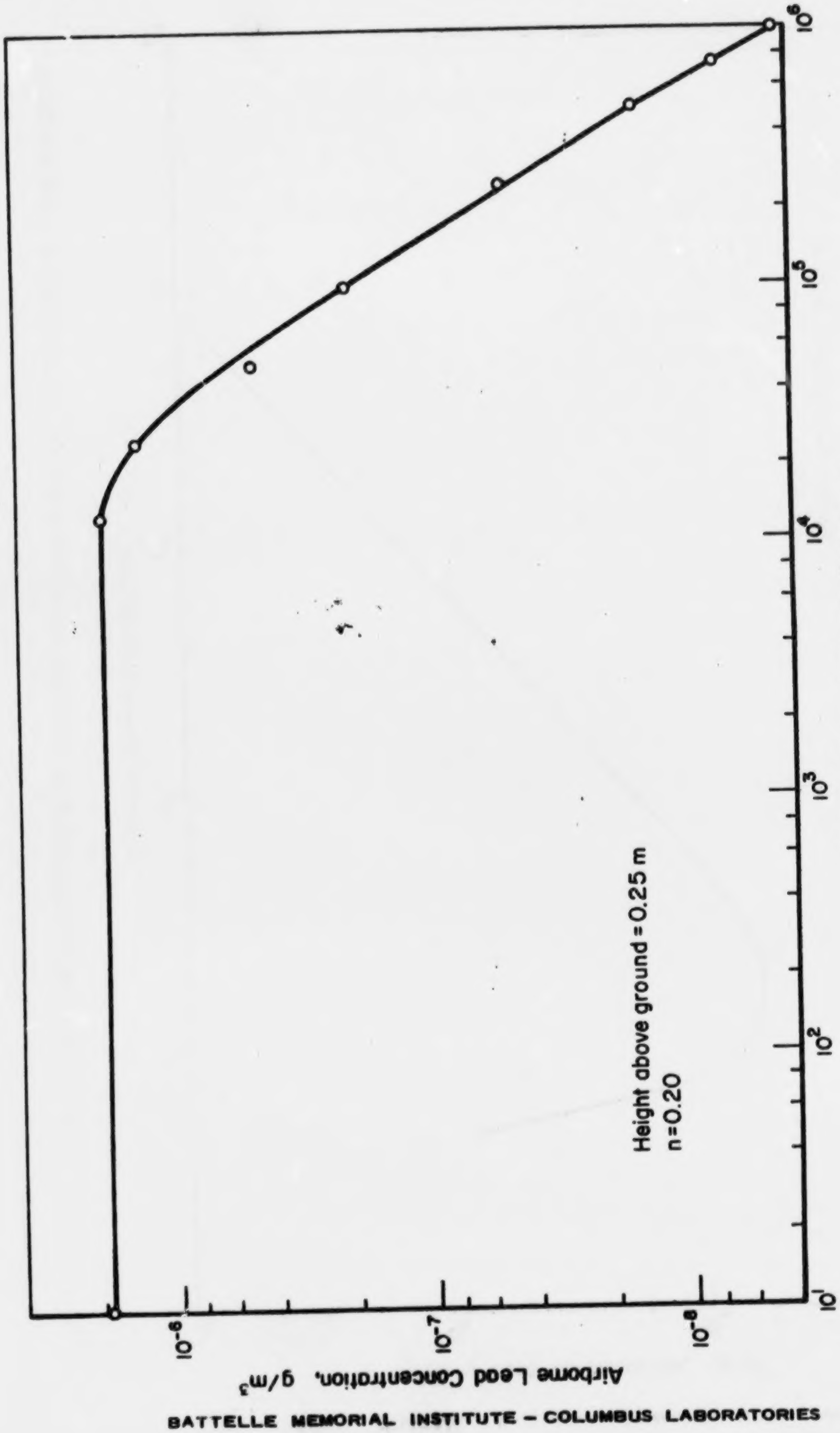


FIGURE 13. AIRBORNE LEAD CONCENTRATIONS IN CINCINNATI DOWNWIND OF AN AREA SOURCE

The airborne lead concentrations for the three types of sources were determined for individuals who were assumed to be inhaling air at one of several locations; the geographical center of Cincinnati, the northern city limits, 12 km north of the city limits, and 87 km north of the city limits. These locations were arbitrarily selected to indicate variations in airborne lead concentrations for individuals who reside at varying distances from the sources. These airborne lead concentrations are listed in Table 17.

With airborne lead concentrations determined for individuals residing at the specified locations in the Cincinnati area and for the three types of sources, the blood lead concentrations due to inhalation of air by these individuals were calculated and are listed in Table 17. In the calculation of the blood lead levels listed in Table 17, it was assumed that in addition to inhaling air in which the airborne lead concentration corresponds to the location of the individual, with respect to the particular source, the individual was also ingesting an average quantity of 300 μg lead per day as reported by Kehoe(55). Due to lack of data which might indicate a nonlinear assimilation of lead into the blood via both the inhalation or the ingestion modes of intake, during higher rates of intake, a linear assimilation of lead into the blood was used for the mathematical model. This linear response fits the available data for lead inhalation studies up to approximately 10 $\mu g/m^3$, however, at higher airborne lead concentrations the predicted blood-lead levels are higher than would be expected.

The blood-lead levels listed in Table 17 were calculated assuming that the airborne lead concentrations were either all soluble forms of lead or all insoluble forms of lead. Since the airborne lead that is inhaled by an individual is most probably insoluble in form, the blood-lead concentrations due to the soluble form have been placed in parentheses. The difference in value of blood-lead concentration as a result of the soluble or insoluble form of the airborne lead upon inhalation is due to the different pathways that the soluble and insoluble forms of lead follow while moving fron the lungs to the blood.

For an individual who is an "average" city dweller, inhaling lead at a constant airborne concentration of 1.0 $\mu g/m^3$ and ingesting 300 μg of lead daily via foodstuffs, the predicted blood-lead level, from calculations using the mathematical model, is 17.6 μg/100 g blood. For an "average" rural dweller ingesting the same daily quantity of lead via foodstuffs and inhaling lead at a constant airborne concentration of 0.5 $\mu g/m^3$, the predicted blood-lead level from calculations using the mathematical model, is 16.5 μg/100 g blood. These predicted blood levels are listed in Table 16. If the intake of lead to an individual via the inhalation mode of intake was completely eliminated, the prediction of the equilibrium blood lead concentration from ingesting 300 μg of lead daily is 15.4 μg/100 g blood. Figure 14 shows the decline of lead in the blood as a function of time if the inhalation of lead is completely eliminated.

Investigation of the ingestion mode of intake at above "average" intake rates of lead by ingestion can readily be done by utilizing the mathematical model. People, and in particular, young children, having pica may ingest abnormally large amounts of lead by periodically ingesting particles of lead based paint. A few small chips of such paints may contain 100 mg or more of lead(28). If an individual that had pica were assumed to chronically ingest 100 mg lead/day, as a result of eating lead based paint chips daily, the predicted response of the blood lead level to this large quantity of lead intake is shown in Figure 15. After one year of this daily intake of lead per day, if the individual stops eating lead based paints and consumes only normal foodstuffs containing lead so that his lead intake is reduced from 100 to 0.3 mg per day, the individual's blood lead

TABLE 17. AIRBORNE LEAD CONCENTRATIONS AT SELECTED LOCATIONS IN CINCINNATI AREA

	Source Type					
	Area		Downwind Line		Crosswind Line	
Location[a]	Airborne Lead Concentration, μg/m³	Blood-Lead Concentration,[b] μg/100 g blood	Airborne Lead Concentration, μg/m³	Blood-Lead Concentration,[b] μg/100 g blood	Airborne Lead Concentration, μg/m³	Blood-Lead Concentration,[b] μg/100 g blood
Center point of city 1	1.9	19.6 (22.4)	13.6	46 (66)	13.2	45 (64)
City limit (north) 2	1.9	19.6 22.4	13.6	46 (66)	0.2	15.8 (16.1)
12 km north of city limits 3	1.4	18.4 (20.4)	0.5	16.5 (17.2)	0.07	15.5 (15.6)
87 km north of city limits 4	0.2	15.8 (16.1)	0.02	15.4 (15.5)	0.02	15.4 (15.5)

(a) Locations of individuals shown in Figure 3.

(b) Blood-lead concentrations enclosed in parentheses are due to airborne levels of lead assumed to be all soluble. Those not in parentheses are due to airborne levels of lead assumed to be all insoluble. These blood-lead concentrations include ingestion of 300 μg lead per day.

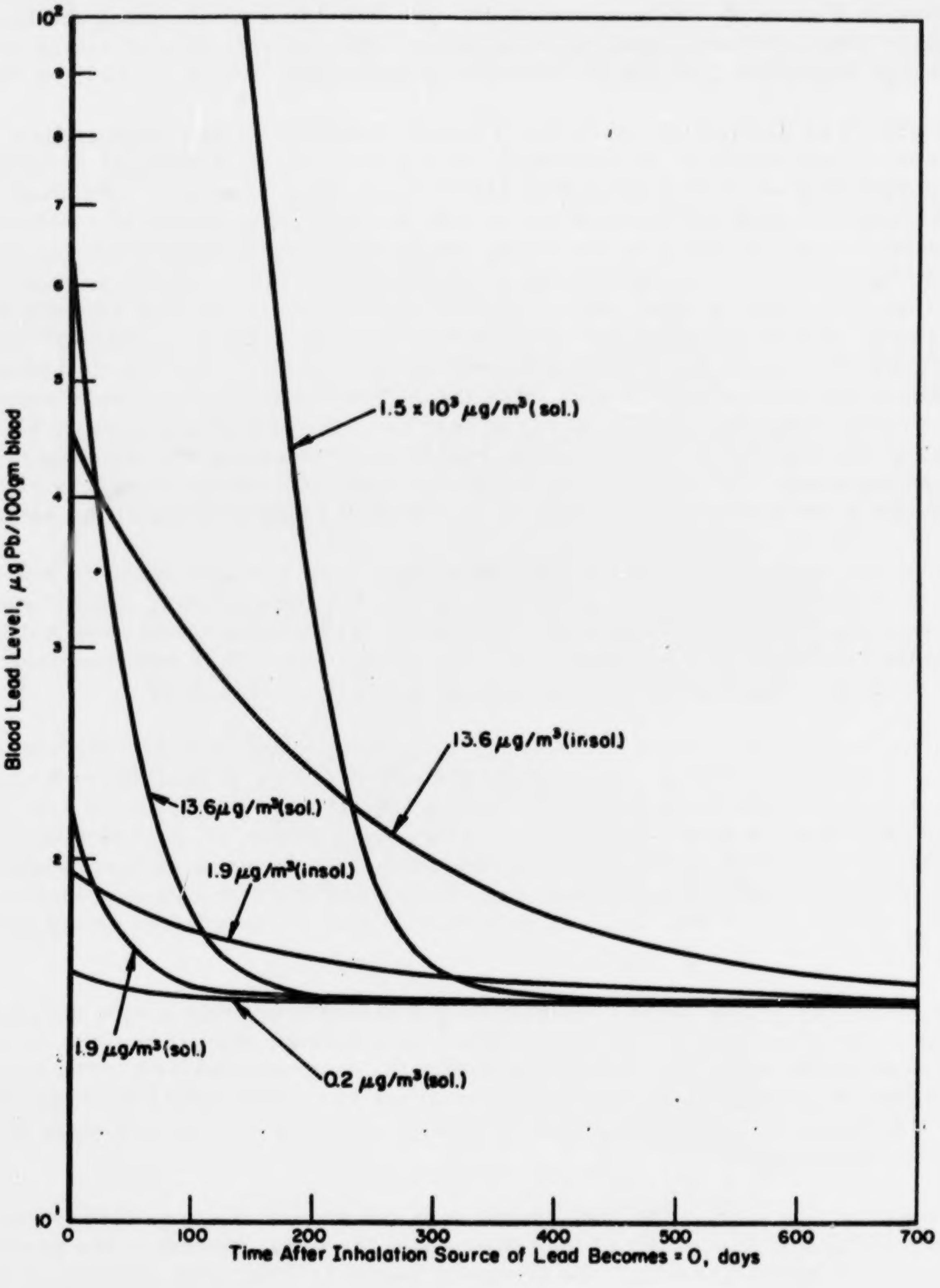


FIGURE 14. BLOOD LEAD CONCENTRATIONS AS A RESULT OF REMOVING LEAD FROM AIR

At time = 0, blood lead level due to continuous daily ingestion of 300 μg Pb per day and inhalation of airborne lead as indicated on each curve.

concentration will begin to decline. The elimination response of lead in blood levels is also shown in Figure 15. It is assumed that this individual while eating the leaded paint particles is also constantly inhaling an airborne lead concentration of 1.0 μg/m^3 as well as ingesting foodstuffs from his normal diet which contain 300 μg of lead per day.

It should be pointed out again that a linear response of lead assimilation into the blood, due to lead intake by an individual, is assumed in the mathematical model as no data are available which can be used to model a nonlinear response. Although the general trend of uptake and elimination of lead in the blood is shown by predicted blood-lead levels obtained by utilizing the linear response of lead assimilation into the blood, the predicted maximum concentrations of lead in blood, due to above "normal" inhalation or ingestion rates of lead, will tend to be higher than levels that actually occur due to this linear response. When and if the data become available, a nonlinear response for assimilation of lead into body organs will be incorporated into the mathematical model since one reference(21) which cites the APHA Committee on Lead Poisoning(62) states, "Values approximating 0.50 mg of lead per 100 g blood are unusual but may occur when the exposure is quite severe; higher concentrations are encountered only in very rare instances of overwhelming accidental exposure; values in excess of 1.0 mg of lead per 100 g blood must be regarded as evidence of contamination of the sample."

An acute ingestion of a large quantity of lead from a single intake in which the individual is assumed to ingest 1 gram of lead which is immediately assimilated into the blood is represented in Figure 16. The blood-lead concentration eventually is reduced to the "normal" concentration of 15.6 g/100 g blood. It is assumed that this individual is also ingesting foodstuffs containing 300 μg of lead daily.

In the case of an individual chronically ingesting more lead than the average intake, e.g., ingesting 1000 μg of lead daily instead of 300 μg of lead daily, and residing in a city location where he is inhaling airborne lead at a concentration of 1.0 μg/m^3, the blood-lead concentration would rise to a new equilibrium of approximately 54 μg/100 g blood as shown in Figure 17. Other combinations of quantities of lead inhaled and/or ingested may be investigated by exercising the mathematical model as a tool to predict an output, in this case blood lead concentrations, that can be utilized as input to the rating system which evaluates projects.

Although the atmospheric transport model does not provide a very detailed representation of the atmospheric diffusion of the stressor lead, the predictions of airborne lead concentrations using this model are within the accuracy desired. The calculated airborne lead concentrations from various sources are within a factor of two of reported values of airborne lead concentrations(32) which is satisfactory at this state of preliminary model development.

Likewise, the lead in the body model does not present a very detailed representation of the biological processes of lead throughout the body, however, the predictions of lead in body organs are within the accuracy desired. The calculated blood lead levels using this model are within a factor of two of reported blood lead levels(32, 54, 55) for inhalation of lead up to airborne lead concentrations several times the normal values or ingestion of lead by eating foodstuffs that are also up to several times the normal values.

Most of the meteorological and biological data required for these models are available in the information data base. The reported values for the data were generally accurate enough (accurate to within a factor of two to ten) for the resolution desired in

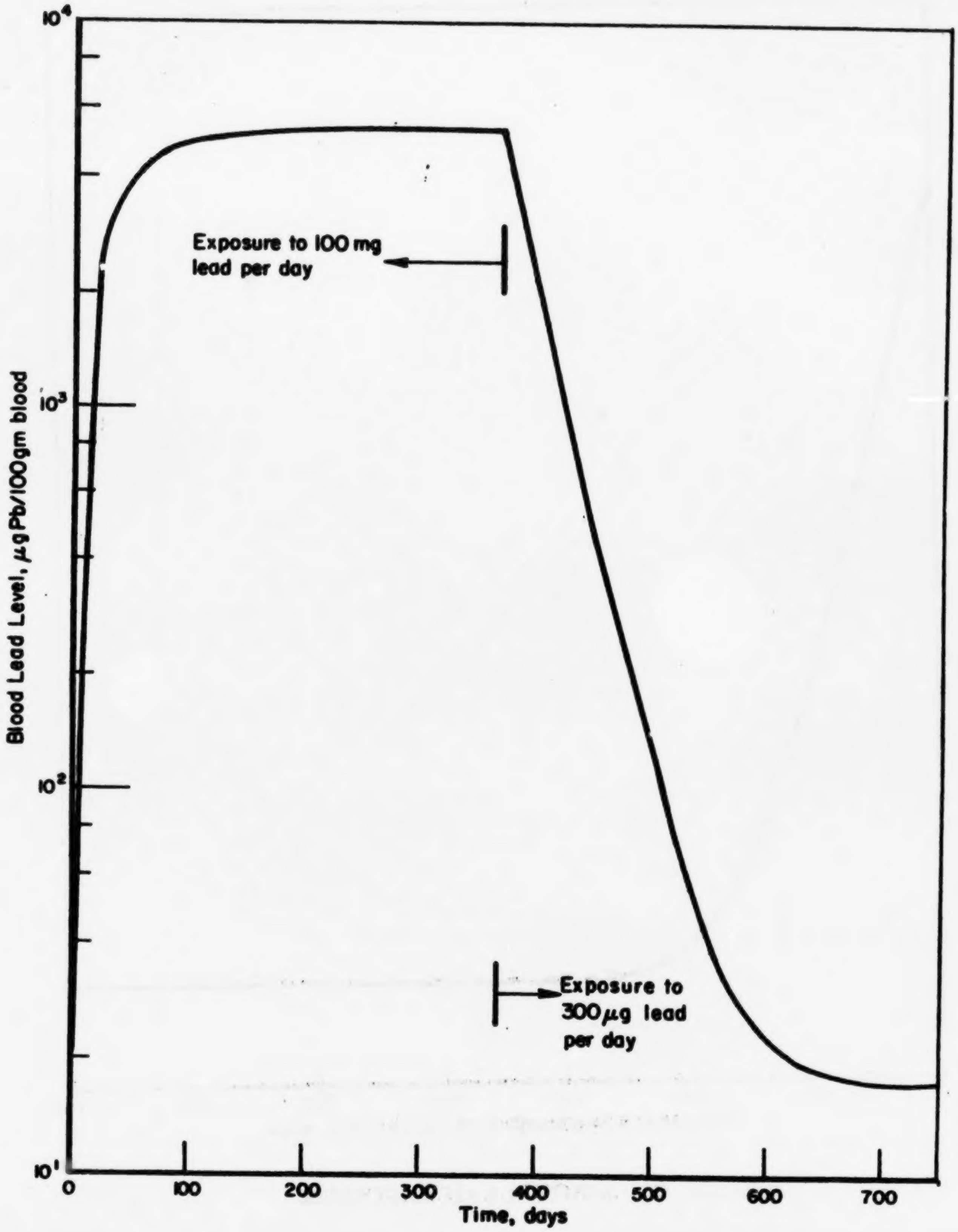


FIGURE 15. CHRONIC LEAD EXPOSURE
Constant inhalation of 1 μg Pb/m^3.

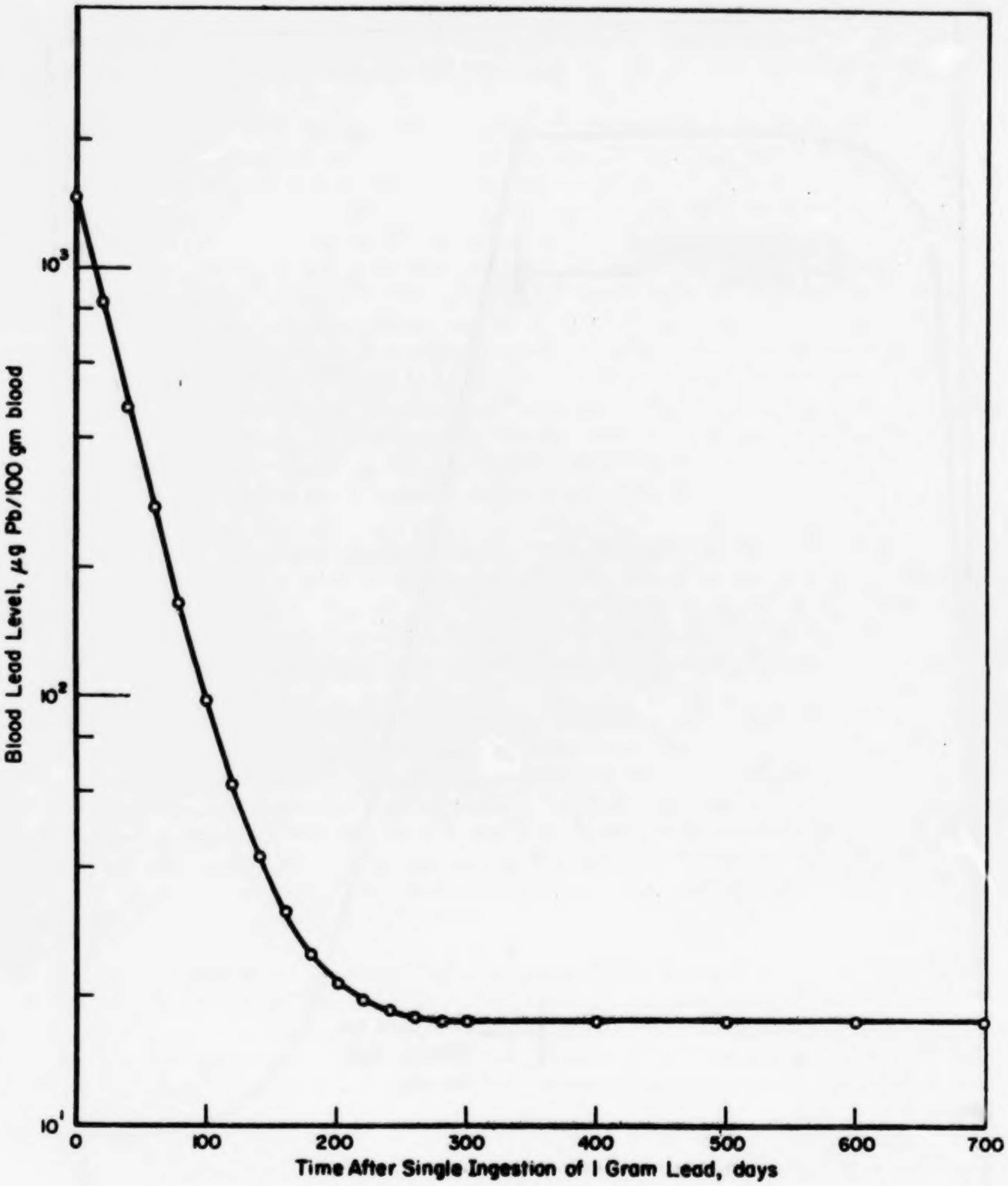


FIGURE 16. ACUTE LEAD EXPOSURE

At T = 0, blood lead level due to single intake of 1 gram lead in addition to continuous ingestion of 300 μg per day and inhalation of 1 μg Pb/m^3.

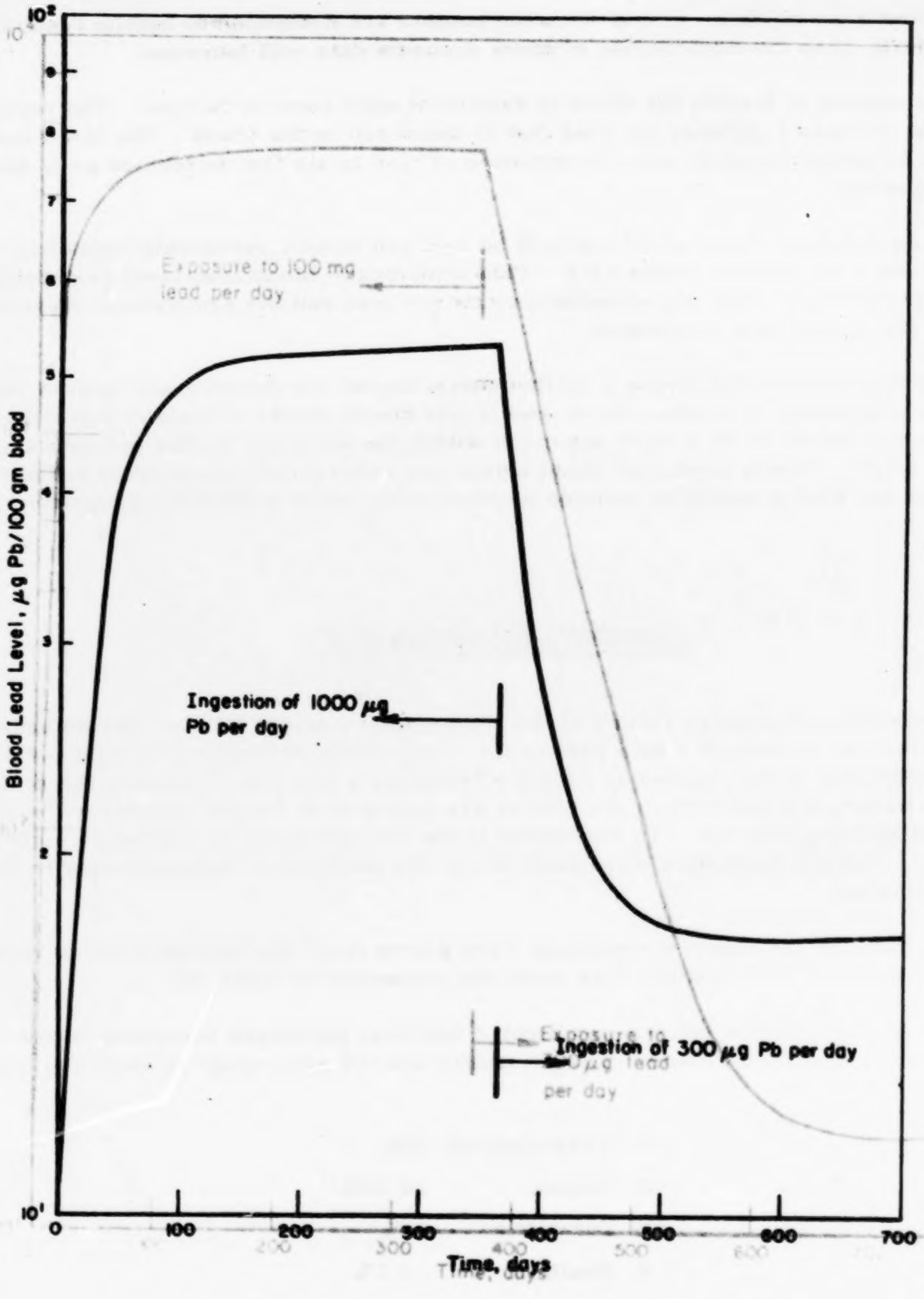


FIGURE 17. CHRONIC LEAD EXPOSURE

Constant inhalation of 1 μg Pb/m^3.

these preliminary models. If and when the models are developed to higher resolution (more detail), then the desirability of more accurate data will increase.

The amount of lead in the blood is dependent upon several factors. The ingestion pathway is the major pathway for lead that is deposited in the blood. The blood lead level will be proportional to the concentration of lead in air that is inhaled or in food that is ingested.

An equilibrium blood lead level will be reached after continuously ingesting or inhaling lead at a constant intake rate. This equilibrium blood lead level is dependent upon the assimilation rate and elimination rate for lead and the equilibrium level will change if the intake rate is changed.

The approach of developing a mathematical model for the stressor lead in order to be able to quantitatively predict blood lead levels due to intake of lead by ingestion or inhalation was shown to be a valid approach within the accuracy of this preliminary modeling effort. These predicted blood levels can subsequently be utilized in the rating of projects for R&D planning to provide a quantitative basis to the R&D program planning.

URGENCY-PICA EXAMPLE

The subject of urgency as one of the elements of a priority index was discussed in general terms in Volume I as a part of the comprehensive planning system for EHS. Data or estimates of the number of people affected as a function of time by the stressor and of the nature and severity of the effects are required to derive values for the urgency over some planning horizon. To determine if the concept could be applied to a problem of interest, urgency estimates were derived for the problem of lead poisoning in children having pica.

The data for the urgency estimates were drawn from the summary of information presented earlier. The specific data used are presented in Table 18.

The total population at risk was divided into four partitions according to the severity of the exposure to lead. The partitions and the percentage in each was set as follows:

(1)	Unrecognized	25%
(2)	Illness	24.95%
(3)	Disability	50%
(4)	Death	0.5%

For each of the partitions it was necessary to develop a worth factor, w_k ($k = 1, 2, 3, 4$). To obtain the maximum utility of the urgency function in priority setting, the worths should be directly related to a cost for the severity of the effect. A rigorous derivation of the costs including indirect costs was not performed but worth factors were assigned that were approximately proportional to the direct costs. Sample calculations were:

(1) The fraction of population in most susceptible age group (i.e., 1 through 3-1/2 years) is constant with time at 5 percent of total population.

(2) The fraction of population in the slum/ghetto area remains contant at 15 percent.

(3) The fraction of slum/ghetto children eating lead remains constant at 20 percent.

(4) The fraction of susceptible housing decreases linearly to zero in 20 years with a housing improvement plan*.

(5) The case of no housing improvement plan was also considered.

(6) The starting point would be the population in 1969 with a housing factor of 1.

Population data including growth rate estimates were taken from Kahn and Wiener**. A low growth rate for the population of 1.2 percent per year over the planning horizon was assumed. The total population at risk, N(t), was calculated using the above assumptions, and the population data and the results are presented. The worth factors used were:

$$w_1 = 10^{-5},\ w_2 = 4,\ w_3 = 20, \text{ and } w_4 = 1.$$

On this basis, the severity function

$$S(t) = \sum_{i=1}^{4} w_i\, N_i\,(t)$$

was calculated for each year. The calculations are shown in Table 18.

Since most of the assumptions are of a linear nature the discrete values of the function S(t) were plotted vs. time and the function was approximated by a straight line defined by the equation

$$S(t) = 348\left(1 - \frac{t}{21}\right) \times 10^3,\ 0 \leq t \leq 20.$$

The priority index U(T) can now be easily calculated for different planning horizons. (It is not necessary to approximate S(t) by a polynomial since the integration can be performed numerically.)

$$U(T) = \frac{1}{T}\int_0^T S(t),\ dt\ \ ;\ 0 \leq t \leq 20;\ T \leq t = 348\left(1 - \frac{T}{42}\right) \times 10^3\ .$$

---

* This figure was broadly drawn from Appendix B, "Rates of Land Utilization for New Apartment Housing in New York, Los Angeles, and Hartford", in The Future of Old Neighborhoods, by B. J. Frieden, MIT Press (1964). The time to rebuild all deteriorated housing in New York ranged from 7.9 to 9.8 years. Estimates for rebuilding in Los Angeles were 14.0 years and for Hartford were 26.1 to 36 years. A linear decrease in susceptible housing was assumed for convenience.

** Kahn, H., and Wiener, A. J., The Year 2000, Macmillan Co., New York, (1967) p 157.

For 5-year intervals:

T	5	10	15	20
$U(T) \times 10^3$	297	265	223	182

The calculations were also made on the assumption that there was no program for inprovement of housing and the current housing situation would remain constant. For this case

$$S(t) = \left(\frac{11}{10}\right)t + 348) \times 10^3$$

and

$$U(T) = \left(348 + \frac{11}{20}\,T\right)$$

For 5-year intervals:

T	5	10	15	20
$U(T) \times 10^3$	351	354	357	360

The two priority indices calculated in this example provide a relative comparison between a housing improvement program and a no-housing improvement program. Although the absolute values of the numbers have little significance, the trends and the divergence are quite noticeable based upon the planning assumptions. However, the prime purpose of this priority ranking system is to compare different environmental hazards. Thus, other hazards which have human health effects should be analyzed in the same manner and then the hazards can be ranked according to the relative priority indices.

TABLE 18. ACUTE LEAD POISONING IN CHILDREN

With Housing Improvement

t	$N(t)$ $\times 10^{+3}$	$N_1(t)$ $\times 10^{+3}$	$N_2(t)$ $\times 10^{+3}$	$N_3(t)$ $\times 10^{+3}$	$N_4(t)$ $\times 10^{+3}$	$S(t)$ $\times 10^{+3}$
1969	30.6	7.65	7.6347	15.3	0.0153	336.5
1970	29.4	7.35	7.3353	14.7	0.0147	323.3
1971	28.2	7.05	7.0359	14.1	0.0141	310.2
1972	26.9	6.725	6.71155	13.45	0.01345	295.9
1973	25.8	6.45	6.4376	12.9	0.0124	283.8
1974	24.5	6.125	6.11275	12.25	0.01225	269.5
1975	23.0	5.75	5.7385	11.5	0.0115	252.9
1976	21.6	5.40	5.3892	10.8	0.0108	237.6
1977	20.3	5.075	5.06485	10.15	0.01015	223.3
1978	--	--	--	--	--	--
1979	17.3	4.325	4.31635	8.65	0.00865	190.3
1981	14.3	3.575	3.56785	7.15	0.00715	157.3
1983	10.8	2.70	2.69460	5.4	0.00540	118.8
1985	7.4	1.85	1.8463	3.7	0.00370	81.4
1987	3.7	0.925	0.92335	1.85	0.00165	40.7
1989	0	0	0	0	0	0
Weights		10^{-5} w_1	4 w_2	20 w_3	1 w_4	

RESEARCH AND DEVELOPMENT PLANNING – AN EXAMPLE

Approach

Since the acute exposure of individuals to lead in the environment has been recognized as a significant health effect and the chronic exposure of individuals to lead in the environment may also produce health effects then a systematic method of program planning directed specifically at this stressor should be followed throughout all organizational levels in EHS as described in Volume I. The information base should be queried to determine what data concerning lead are already available and what types of research concerning lead are currently underway. A schematic diagram of the environmental pathways of lead from source to intake by an individual may then be formulated. This pollution chain diagram may be much more detailed than the available quantitative data that are contained in the information base since the pollution chain diagram is the qualitative representation of lead pathways through the environment. However, the quantitative assessments are limited by the data stored in the information base or to reasonable estimates of missing information.

Utilizing the pathways of lead in the environment, as indicated in the pollution chain diagram, a mathematical model can be developed to simulate the emission of lead by various sorces; transport of lead through various pathways to man; and the intake, deposition, and elimination of lead in man. The output of the mathematical model is a quantitative value of the amount of lead in several body organs. The predicted quantities of lead in these body organs can be investigated as a function of the parameters in the mathematical model, such as, emission rates of lead from sources, rates of transfer of lead through the environment, specific pathways of travel to man, mode of intake to man, etc. In using the model, parameter values which are thought to be conservative, i. e., values which tend to overestimate results, will help compensate for any uncertainties in these parameters so that predicted amounts of lead in body organs will in all cases be equal to or higher than the actual values. However, the model can be developed to simulate real world interactions closely to the extent of available data and knowledge. The predictions of the mathematical model are quantitative and are based upon a systematic utilization of basic data available as compared to guesstimates or intuition. The model can be used to investigate all pathways of lead intake and quantitatively predict lead in body organs from all modes of intake and thus, may determine the most significant pathways for detailed study. Some specific illustrations of the model development efforts have been described in a previous section of this model case study. Of utmost importance is the quantitative output of the model which can be used as a quantitative base for rating the R&D program objectives.

Concurrent with the development of the mathematical model, and dependent upon the information base as well as the pollution chain diagram is the formulation of the set of objectives necessary to develop a systematic approach to R&D project planning for the environmental stressor lead. A set of objectives with corresponding subsets of the tasks and projects necessary to obtain the goals stated by each objective can be formulated by use of supporting data contained in the information base. The R&D program planning to select objectives, tasks, and projects of concern for the stressor lead can be performed at any organizational level, but as described in Volume I, program guidance should initiate at the Service level, detailed plans should be developed at lower levels, and review and integration of the plans should be done at the Service level. With a complete, idealistically planned program outlined for lead it will soon be evident that all of the listed projects cannot be accomplished due to limitations of economics, manpower, facilities, time, etc. Thus, the projects must be evaluated systematically in order to determine for the total problem of lead in the environment the priority of each project so that the projects may each be ranked in an order of importance requiring action to be taken. A ranked-order of projects which concern the total problem of the quality of life of man in his environment as affected by the stressor lead, lends a logical method for selection of the proper projects for allocation of funding, based upon a systematic appraisal of all pertinent projects and subsequent determination of which projects will contribute the most to the problem (as set by priorities).

Determining the priorities for projects, which allows a subsequent ranking profile for these projects, is based upon being able to systematically appraise the projects. The predicted-quantitative output of the model is the basis for the subsequent rating of projects which involves the determination of the severity of the effect and the urgency for action (both elements of the rating system) from each pathway of lead that is of concern. The lead pathways may correspond to specific objectives in the R&D program planning for the stressor lead, for example, an objective concerning children who have pica would correspond to the ingestion pathway for lead.

The Example for Lead

For the case study of the environmental stressor lead, it has been assumed that lead has been recognized as a significant health problem, therefore an R&D program must be planned specifically for the stressor lead. The data that were gathered for the information base were investigated in order to formulate a pollution chain diagram and to develop a mathematical model for lead. Concurrent with the diagram and the model the information base was inspected in order to establish a set of objectives so that a systematic approach could be followed for R&D project planning for the stressor lead. A listing of several objectives based upon a study of available information concerning the stressor lead is given in Table 19. These six objectives are not listed in order of importance and the list is not all encompassing. However, the statement of these objectives is the initial step to R&D project planning for the stressor lead from which the alternatives, tasks, and projects can subsequently be established.

TABLE 19. OBJECTIVES FOR R&D PLANNING FOR THE STRESSOR LEAD[(a)]

(1) Eliminate lead poisoning in urban ghetto children having pica
(2) Eliminate lead from bootleg liquor
(3) Minimize and control occupational exposures
(4) Minimize body burden of lead in the general population
(5) Identify population groups sensitive to lead exposure
(6) Determine the effects of chronic lead exposure

(a) It has been assumed for this example that lead in the environment has been recognized as a significant health problem, therefore we must do something.

Again utilizing the information gathered for the lead case study, a set of alternatives for each stated objective can be established. These alternatives are the methods which may be followed to accomplish the statement of the objective and are listed in Table 20. The development of lists of tasks and projects for each alternative can be done similarly. Tables 21 and 22 list the tasks and projects, respectively for only three of the stated objectives in order to show some examples of the establishment of a list of projects which should ultimately be completed. These projects can provide the required input data to the tasks and alternatives to determine which are most vital to meeting the statement of the objectives. (A listing of alternatives for each objective, a list of tasks for each alternative, and a list of projects for each task would be too large a list to present in this example.)

In addition to the scientific and technical information presented on lead earlier in this volume, R&D project planning requires a knowledge of current research projects related to lead in the environment. A survey of EHS projects was made through the KWOC index described in Volume II. This survey includes information on NAPCA projects and all grants active within CPEHS in February, 1969, and the results are tabulated in Appendix A for reference. A list of notices on research projects registered with the Science Information Exchange of the Smithsonian Institution was requested as a survey of current research outside of EHS. The results of this survey are also included in the Appendix. Since the purpose of the model case study was only to exercise information networks, and not to develop comprehensive R&D plans, no attempt was made to eliminate from the list of needed research projects those that are currently being conducted.

The discussion of project rating systems was discussed in detail in Volume I. The mathematical model for the environmental stressor lead provides the initial input to the rating system. Thus, this model is a useful tool in R&D project planning. The predictive output of the model, is calculated under present environmental conditions, i. e., the blood lead level, as a result of lead intake can be determined for any modes and rates of lead intake. These blood lead levels are required for evaluation of the alternatives to the objectives. For example, the first objective which is elimination of lead poisoning in urban ghetto cheldren having pica has six stated alternatives to meet the objective. The mathematical model for lead can quantitatively predict the effect upon blood lead levels in ghetto children having pica over future time periods as a result of implementation of each alternative. Thus, a quantitative measure of blood lead levels in the ghetto children is used as an input to the rating system to compare alternatives instead of using only an intuitive judgment.

The mathematical model developed for lead previously described in this model case study is a lower-echelon model in the hierarchial modeling approach which is discussed in detail in Volume I. This lead model yields quantitative predictions even though the model is not comprehensive. Other lower-echelon models which may be incorporated into this hierarchy of models, e. g., economic models, pathway models, biological models, political models, educational models, etc., may only be qualitative in nature. In fact, models of other stressors may, due to lack of existing data, be only qualitative in nature until the pertinent data are obtained and generated for use as input data so that the models can yield quantitative results. The pollution chain diagram is in a sense a qualitative model.

TABLE 20. ALTERNATIVES FOR R&D PLANNING FOR THE STRESSOR LEAD(a)

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1. 1 Remove and repaint interior surfaces in houses and buildings
1. 2 Education
*1. 3 Screening and early detection of lead in children
1. 4 Renewal of urban housing
1. 5 Overcoat leaded painted surface
1. 6 Reduce lead in exterior paints to safe level.

2. 1 Eliminate bootleg liquor
2. 2 Education
2. 3 Eliminate socio-economic factors
*2. 4 Screening and detection in alcoholics.

*3. 1 Review and elimination of hazardous conditions in occupational environments
3. 2 Training of workers
3. 3 Enforce standards.

4. 1 Control lead emission
    4. 1. 1. Gasoline standards
        *4. 1. 1. 1. Eliminate lead in gasoline
        *4. 1. 1. 2. Exhaust control
    *4. 1. 2. Industrial standards
4. 2 Control lead ingestion levels
    *4. 2. 1. Drinking water
    *4. 2. 2. Food
*4. 3 Control lead inhalation
*4. 4 Maximize elimination rates.

5. 1 Epidemiological studies
    *5. 1. 1. Lead workers
    *5. 1. 2. Other population groups.

*6. 1 Animal studies
*6. 2 Trace metals in humans
*6. 3 Epidemiological studies.

(a) All alternatives preceded by an asterisk are in R&D category. Alternatives not preceded by an asterisk are in program categories other than R&D.

TABLE 21. TASKS FOR R&D PLANNING FOR THE STRESSOR LEAD[a]

1.11	Locations of homes where lead-based paints are used
*1.111	Determination that lead-based paints are used in homes.
*1.12	Develop optimum economical methods for repainting and replacing
1.21	Educate the educators
1.22	Develop training literature for various groups: parents, social workers, government officials, teachers, etc.
1.23	Mass-media communication: radio, television, newspapers
*1.31	Develop screening methods and techniques
*1.32	Develop screening program
1.33	Follow up programs
*1.34	Therapeutic approaches
1.41	Survey of fringe area homes after urban renewal
*4.111	Research to establish airborne lead concentrations based upon health effects
*4.121	Research to establish airborne lead concentrations based upon health effects
*4.212	Investigate lead concentrations and lead-removal techniques in drinking water
4.213	Surveillance of lead in water
*4.221	Same as 4.211
*4.222	Investigate lead concentrations in food and removal of lead sources in food
4.223	Surveillance of lead in food
*4.224	Set food standards for lead
*4.31	Research to control sources
4.32	Set source emission standards
4.33	Wear respirators
*6.11	Research with several types of animal species to determine chronic somatic and genetic effects of lead, using various lead compounds, lead concentrations, particle size, etc., via all modes of exposure
*6.12	Research to investigate possible synergistic effects of lead
*6.21	Research of trace metals in cadavers
*6.22	Research of trace metals in animals
*6.31	Epidemiology of lead workers
*6.32	Epidemiology of selected communities
*6.33	Epidemiology of a total community

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[a] All tasks preceded by an asterisk are in R&D category. Tasks not preceded by an asterisk are in program categories other than R&D.

TABLE 22. PROJECTS FOR R&D PLANNING FOR THE STRESSOR LEAD[a]

*1.111	Establish field method for detection of lead in paint
*1.112	Establish survey method to include all homes which may have lead based painted walls
*1.121	Establish laboratory program to develop more economical methods to repaint walls
*1.122	Establish laboratory program to develop more economical methods to replace walls
*1.211	Establish training courses for physicians, health and safety officials, teachers, employers, etc.
*1.212	Establish and staff training centers
1.222	Publish training literature for various groups
1.223	Disseminate literature to the various groups
1.231	Publish literature for use by mass media
1.232	Disseminate literature to the various groups
*1.311	Establish laboratories for screening operations
*1.312	Establish reliable laboratory techniques for screening
*1.321	Establish method to obtain screening of all young children
*1.322	Set limiting screening criteria for further investigation of subjects
*1.331	Establish methods for follow up screening
*1.341	Establish methods for medical treatment following positive lead levels
*1.342	Establish medical facilities for therapy
*1.343	Establish program to reexamine subjects
*1.411	Establish comprehensive survey methods
*4.1111	Establish allowable levels of lead in gasoline
4.1112	Manufacture gasoline with lead within allowable levels
*4.1121	Establish allowable levels of lead in exhausts
*4.1122	Develop exhaust control devices for lead
4.1123	Manufacture exhaust control devices
*4.1124	Establish inspection procedures and standards for exhaust control devices
*4.2111	Establish procedures to remove lead from drinking water to below allowable levels
*4.2121	Establish sensitive method of measuring lead in water

TABLE 22. (Continued)

*4.2122	**Establish methods to remove lead sources from drinking water supplies**
*4.2131	**Establish surveillance procedures in drinking water**
*4.2211	**Establish allowable levels of lead in food**
*4.2221	**Establish sensitive method of measuring lead in food**
*4.2231	**Establish surveillance procedures in food**
*4.2241	**Establish human health effects due to ingestion of food**
*4.2242	**Establish standards for lead in food**
4.2243	**Publish standards for lead in food**
*4.311	Establish inventory of all sources
*4.312	Establish methods for source control
4.313	Manufacture source control equipment
*4.314	Establish inpsection procedures
*4.321	Establish the human health effects due to varying levels of atmospheric lead
*4.322	Establish criteria for atmospheric lead concentrations based upon health effects
*4.331	Establish respiratory control methods
4.332	Manufacture respirators
4.333	Disseminate respirators and pertinent information on respirators use to general population
*6.111	Establish laboratory program to perform animal studies
*6.112	Establish laboratory facilities and staff to conduct animal studies
*6.121	Establish methods to determine synergistic effects
*6.122	Establish laboratory programs to implement synergistic studies
*6.211	Establish program to measure lead in cadavers
*6.212	Establish program to measure lead intakes and elimination rates in humans
*6.221	Establish program to measure lead intake, deposition and elimination from animals
*6.311	Establish program to document epidemiology of lead workers
*6.321	Establish program to document the lead epidemiology of selected communities
*6.331	Establish program to document the lead epidemiology of the total community

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(a) All projects preceded by an asterisk are in R&D category. Projects not preceded by an asterisk are in program categories other than R&D.

Thus, the R&D project planning for lead begins with quantitative predictions generated by exercising the mathematical model developed for lead. This quantitative output, through the rating system, yields a quantitative comparison of the alternatives available to meet the stated objectives. The R&D project planning from the level of tasks to projects, however, must at this time be more qualitative than quantitative since the models required for quantitative judgments are still mostly in the qualitative stage of development in this project.

As an example, to attempt to illustrate the preceding discussion, the objective to eliminate lead poisoning in ghetto children having pica has six stated alternatives. The blood lead concentrations as a result of investigating each alternative, individually, can be predicted by exercising the mathematical model with the proper data that is representative of each alternative. As discussed in Volume I, the urgency and severity functions expressed as time varying functions extended to future times, e.g., 5 to 20 years, can then be calculated for these six alternatives utilizing the predicted blood lead levels for the respective alternatives.

The degree of confidence may not be high at this time for a quantitative approach to R&D planning for the tasks and the projects. However, with more comprehensive development of the mathematical model the degree of confidence increases for quantitative assessments. Even at this state of model development some quantitative assessments can be made. For the alternative of screening and early detection in young children, for the ghetto children-pica objective, it may be determined by simulation that one ethnic-age group is significantly more sensitive to the stressor lead than others and should be examined in more detail for blood lead concentration than other ethnic-age groups. These predictions can only become quantitative when the information base becomes extensive and the model becomes more comprehensive than at present. The several tasks of (a) developing screening methods and techniques, (b) developing screening programs, and (c) follow-up programs may all be modified due to the determination of a sensitive ethnic-age group, e.g., the screening methods, screening program, and follow-up programs could now concentrate mainly on the sensitive ethnic-age group. Furthermore, at the project level the quantitative assessment of a sensitive ethnic-age group would probably modify the project of screening all young children to a project of screening mainly the sensitive ethnic-age group.

Project Performance Measure

The R&D project planning must ultimately make a comparison of the list of projects for the stressor lead that are compiled so that allocation of funding for these projects may proceed on a systematic basis. The projects should be compared to determine which projects are judged to have the greatest effect in fulfilling the statement of the objectives for lead.

Use of a performance index, PI, in the rating of various alternative projects has been described in Volume I. To provide an illustration of the approach, performance indices were developed for several of the projects identified in this lead model case study. The method used is similar to that followed in DARE(63) or described by Churchman and Ackoff(64). The six objectives in Table 19 were first ranked in order of importance. Then pair-wise comparisons among the objectives were made. A quantitative factor was assigned to each objective which reflected the ratio of its importance to the one immediately below it in the list. A value of unity was arbitrarily assigned to the sixth objective. Comparisons among the objectives were terminated with this simple pair-wise comparison which is adequate to illustrate the method. However, a more rigorous application of the technique would require grouping of the objectives and further comparison to be certain that the values for all possible combinations were also in the proper order. The ratios between the objectives were converted into weights by taking products from the bottom of the list to the top, i. e., the weight of the fifth objective was found by multiplying the ratio of the fifth to the sixth by unity, the weight of the fourth objective was determined by multiplying the weight of the fifth objective by the ratio of the fourth to the fifth, etc. The weights were then normalized so that their sum equal 1.0. The results of the various steps to obtain the weights of the objectives are shown in Table 23.

TABLE 23. OBJECTIVE WEIGHTS

Objective[(a)]	Pair-Wise Ratio of Objectives	Weights	Normalized Weights
1. Eliminate lead poisoning in urban ghetto children having pica	5	480	0.745
2. Minimize body burden of lead in the general population	2	96	0.149
3. Determine the effects of chronic lead exposure	3	48	0.074
4. Minimize and control occupational exposure	4	16	0.025
5. Identify population groups sensitive to lead exposure	4	4	0.006
6. Eliminate lead from bootleg liquor	1	1	0.001
		$\Sigma = 645$	$\Sigma = 1.000$

(a) Objectives from Table 19.

Worth assessments were then made for each of the projects under consideration. This was done in a subjective manner by assigning a value between zero and one to each project as it related to each objective. For example, if the project under consideration was expected to accomplish about 90 percent of a given objective, a worth of 0.9 would be assigned to it. The worths for the projects considered are tabulated under the six objectives in Table 24.

The probability of being able to accomplish the project was then estimated. This probability depends upon the existing state of knowledge on understanding of the area under consideration with respect to the objectives. Values between zero and one were assigned to the probability of success. An estimate of the cost of the project was also made. Since the cost can be the dominant factor in the performance index, five arbitrary cost categories were used in this example. These were < $\$1 \times 10^5$. $\$1 \times 10^5$ to $\$2.5 \times 10^5$, $\$2.5 \times 10^5$ to $\$5.0 \times 10^5$, $\$5.0 \times 10^5$ to $\$1.0 \times 10^6$, and > $\$1 \times 10^6$ with a cost

TABLE 24. WORTH ASSESSMENTS

Project[(a)]	Objectives 1	2	3	4	5	6	M_p
1. Establish laboratory program to develop more economical methods to repaint walls	0.3	0.05	0	0	0	0	0.23
2. Establish laboratories for lead screening operations for children	0.1	0.05	0.1	0	0.1	0	0.09
3. Establish allowable levels of lead in gasoline	0	0.2	0	0.05	0	0	0.30
4. Establish allowable levels of lead in automobile exhaust	0	0.2	0	0.05	0	0	0.30
5. Establish methods to determine synergistic effects	0.1	0.1	0.1	0.1	0.05	0	0.10
6. Establish program to document the lead epidemiology of selected communities	0.1	0.05	0.2	0.1	0.3	0.1	0.10

(a) These projects are several arbitrarily selected from Table 22.

value assignment of 1, 2, 3, 4, 5, respectively. In this example, only approximate estimates of project costs were made but in practice it is expected that the detailed cost estimates would be provided by the proposer of the project. Even though better cost estimates would be available in this case, it still is desirable to group them by categories. Table 25 summarizes the factors for the probability of success and the costs for the projects considered in this example.

The final performance index $(PI)_p$, for each project p was calculated from the following relationship

$$(PI)_p = \frac{M_p \times P}{C}$$

where

$$M_p = \sum_{i=1}^{6} w_i E_{ij}$$

w_i = weight of the i^{th} objective

E_{ij} = worth of the j^{th} project with respect to the i^{th} objective

P = probability of success

C = cost of the project.

The performance factors are given in Table 26. It would be emphasized at this point that these performance factors should be used to guide the decision maker and are not to be accepted at face value as an absolute measure of project performance. The general trends are probably valid but to conclude that a given project is worth x times more than another is not justified.

TABLE 25. PROBABILITY AND COST

Project[(a)]	Probability of Accomplishing Project	Cost of Accomplishing Project
1. Establish laboratory program to develop more economical methods to repaint walls	0.9	1
2. Establish laboratories for lead screening operations for children	0.9	3
3. Establish allowable levels of lead in gasoline	0.9	4
4. Establish allowable levels of lead in automobile exhaust	0.9	4
5. Establish methods to determine synergistic effects	0.5	5
6. Establish program to document the lead epidemiology of selected communities	0.7	2

(a) These projects are several arbitrarily selected from Table 22.

TABLE 26. PERFORMANCE FACTORS

Project[(a)]	Performance Factor for Project
1. Establish laboratory program to develop more economical methods to repaint walls	0.21
2. Establish laboratories for lead screening operations for children	0.03
3. Establish allowable levels of lead in gasoline	0.07
4. Establish allowable levels of lead in automobile exhaust	0.07
5. Establish methods to determine synergistic effects	0.01
6. Establish program to document the lead epidemiology of selected communities	0.04

(a) These projects are several arbitrarily selected from Table 22.

RECOMMENDATIONS

This model study indicates that environmental sources of lead constitute a major source of lead intake, and lead in the ambient air may not contribute materially to the body burden of lead. Considerable information concerning the toxiciology of pediatric and adult plumbism was appraised. The biochemical, clinical, metabolic, diagnostic, and therapeutic parameters of plumbism have been discussed.

In this model case study the stressor lead was specifically investigated. The relative importance of lead compared to other stressors was not evaluated. Thus, the following recommendations are made to implement environmental health criteria concerning lead, based only upon this model case study for lead without the relative importance determined at this time of lead to other stressors:

(1) Conduct research to determine the chronic effects of lead in the atmosphere on health. To date, definitive information on the effects of airborne lead on health has not been obtained.

(2) Lead overexposure can change certain biochemical functions, but techniques have not been developed to employ these changes for biological monitoring. For example, further studies on ALA metabolism could lead to the development of a technique for detecting subtle biochemical changes caused by intake of lead either by inhalation or ingestion. This ALA screening should be developed so that elevated ALA levels would indicate recent intake of higher than normal quantities of lead.

(3) Research should be conducted to develop more effective therapeutic agents for plumbism, as some chelating agents may produce more harmful effects than the stressor itself. This is extremely important in pica therapy and massive overdoses of lead in industry.

(4) Reexamine the effect of shifting technology in the utilization of lead, e.g., the large scale use of lead storage batteries in electric cars. Conceivably, this could lead to a reduction in the environmental burden of lead from gasoline additives. The advantages and disadvantages of this shift should be explored from an environmental health point of view and second or higher order factors should be evaluated.

(5) Synergistic effects. Before the environmental character of lead can be finalized, there is need to determine possible synergistic effects of other stressors. For example, oxides of sulfur, nitrogen, and metals that comprise the environmental burden, need to be investigated.

(6) The preliminary mathematical model developed for this model test case for the stressor lead can be used to quantitatively investigate some of the previous recommendations and used as a tool for R&D planning. The model should eventually be developed into a more comprehensive detailed model which will require better data than is available at the present time. Some of these data are:

(a) Chemical forms of lead inhaled and ingested

(b) Assimilation of lead upon entering the body

(c) Elimination rates by body organs

(d) Particle size distribution of inhaled lead

(e) Emission rates of lead sources

(f) Meteorological data, specifically in mining, reclamation, and manufacturing areas.

The severity of the health and associated economic effects from the ingestion of lead-based paints by children having pica has been recognized. Several large cities have screening programs under way to detect high levels of lead in children. In addition to the several recommendations pertaining to research programs for the stressor lead, the elimination of ingestion of lead by ghetto children having pica is highly desirable, as this mode of intake was shown by the preliminary lead model to yield high lead-body concentrations as compared with other modes of intake. Programs should be initiated to locate and remove lead-based paints from ghetto houses and screen all children who may be expected to have elevated lead-body burdens.

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APPENDIX A

CURRENT R&D PROJECTS – LEAD

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CURRENT R&D PROJECTS – LEAD

Within EHS (NAPCA Projects and All Grants)

A1500-11RD	Lewis, T. T., Dr. Campbell, K. I., Dr. Chronic & Acute Toxicity of Auto Exhaust, Sulfur Dioxide, Nitrogen Oxides and (Lead Particulates) Health Effects Research 1055 Laidlaw Ave., Cincinnati, Ohio
A1526-11RD	Newill, V. A., M. D. Long-Term Health Effects of Lead Arsenate Health Effects Research Program 411 W. Chapel Hill St., Durham, North Carolina 27701 State of Washington (NCDC), Wenatchee, Washington
A1549-11RD	Brown, J., Ph. D. Comprehensive Annotated Lead Bibliography Health Effects Research Program OTIP, NAPCA, Arlington, Virginia University of Cincinnati, Cincinnati, Ohio
A1550-11RD	Horton, R. J. M. Surveillance of Population Lead Levels Health Effects Research Program 1055 Laidlaw Avenue, Cincinnati, Ohio 45237 Kettering Laboratory, University of Cincinnati, Cincinnati, Ohio, Philadelphia, Pennsylvania, and Los Angeles, California
AP00585-02	Winchester, J. W. Composition of Lead Halide Pollution Aerosols University of Michigan, Ann Arbor, Michigan
APOO875-04	Chow, T. J. Lead Isotopic Chemistry for Pollution Research University of California at San Diego, San Diego, California
UI00399-16	Chisolm, J. J., Jr. Biochemical Effects of Lead Poisoning Johns Hopkins University, Baltimore, Maryland
UI00406-08	Hammond, P. B. Lead Chelation in Rats University of Minnesota, St. Paul, Minnesota

SCIENCE INFORMATION EXCHANGE

Supporting Agency	Title	Principal Investigator/Institution	Period
Agriculture Department, Agricultural Research Svs.	Behavior of Pesticides and Pollutants in Soil, Water, and Plants	A. W. Taylor U. S. Dept. of Agriculture Beltsville, Maryland	7/68 to 6/69
Arizona State Govt.	Trace and Tracer Elements in Ground Water: Phase III	G. R. Dutt University of Arizona Tucson, Arizona	7/68 to 8/68
	Trace and Tracer Elements in Ground Water	G. R. Dutt University of Arizona Tucson, Arizona	7/68 to 6/69
Atomic Energy Commission, Biology & Medicine Div.	Radioactive Food-Chains in the Subarctic Environment	J. M. Miettinen Helsingin YLI Helsing. Univ. Helsinki, Finland	8/69 to 8/70
Canadian Government, Dept. of Natl. Hlth & Welfare	Continuous Atmospheric Lead Sampler	J. M. Beeckmans University of Western Ontario London – Ontario, Canada	7/69 to 6/70
Commerce Department, Nat'l Bureau of Standards	Polarographic Techniques of Analysis	Unknown U. S. Dept. of Commerce Washington, D. C.	7/68 to 6/69
Interior Department, Bureau of Commercial Fisheries	Water Quality of Tennessee River below Kentucky Dam	H. M. Hancock State Dept. Frankfort, Kentucky	3/69 to 6/69
Office of Water Resources Res.	Automated Potentiometric Techniques for the On-State Monitoring of Ions Important in Water Quality Control	S. E. Manahan University of Missouri Columbia, Missouri	7/69 to 6/70
	Device for Field Determination of Heavy Metals in Natural Waters	R. E. Vanatta Ball State University Muncie, Indiana	9/69 to 9/70
Interior Department, Office of Water Resources Res.	The Impact of Lead-Zinc Mining on the Quality and Ecology of Surface Waters in Southeast Missouri	E. A. Bolter University of Missouri Rolla, Missouri	7/69 to 6/70
	Effect of Surfactant Micelles on Metal Ion Removal by Floration from Metallurgical Wastewater	R. L. Venable University of Missouri Rolla, Missouri	7/69 to 6/70
	Geochemical Effects of Mining Pollution on the Streams of SE Missouri	E. Bolter University of Missouri Rolla, Missouri	7/69 to 6/70
Bureau of Sport Fish & Wildlife	A Study of Suspected Lead Poisoning of Swans and Geese	M. Perry State Div. of Conservation Providence, Rhode Island	10/69 to 9/70

A-3 and A-4

SCIENCE INFORMATION EXCHANGE
(Continued)

Supporting Agency	Title	Principal Investigator/Institution	Period
	Chemical Characterization of Waters of Prairie and Forest Wetlands	V. A. Adomaitis U. S. Dept. of Interior Jamestown, North Dakoth	7/68 to 6/69
Bureau of Mines	Chlorination of Pyrite Wastes	G. W. Elger U. S. Dept. of Interior Albany, Oregon	7/69 to 6/70
	Recovering and Refining Secondary Metals and Alloys	H. S. Caldwell U. S. Dept. of Interior College Park, Maryland	7/69 to 6/70
	Recovery of Metals and Phosphates from Metal Coating Wastes	A. A. Cochran U. S. Dept. of Interior Rolla, Missouri	7/69 to 6/70 terminated
	Interactions Between Fuel Composition and Exhaust Emissions	J. R. Allsup U. S. Dept. of Interior Bartlesville, Oklahoma	7/68 to 6/69
New Jersey State Govt.	Relationship of Lead in the Air, Soil and Plants to Proximity to Highway Traffic	R. H. Daines Rutgers The State University New Brunswick, New Jersey	7/68 to 6/69
University of Texas	The Quantitative Determination of Lead in Lake Water via Atomic Absorption Spectrophotometer	H. D. McAfee University of Texas Arlington, Texas	7/69 to 6/70
Utah State Govt.	Treatment of Tailings to Enhance plant Growth and Promote Stabilization of Tailings	H. B. Peterson Utah State University Logan, Utah	7/68 to 6/69
Utah State Govt.	Lead Shot, Its Settlement, Oxidation, and Availability to Waterfowl	J. B. Low Utah State University Logan, Utah	7/69 to 6/70

APPENDIX B

CATEGORIZED BIBLIOGRAPHY – LEAD

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CATEGORIZED BIBLIOGRAPHY – LEAD

Production/Use Pattern

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a major source of environmental lead. Each gallon of today's gasoline contains on the average of 2 to 3 grams of lead (maximum of 4 grams), which adds up to approximately 540 million pounds of lead consumed, according to gasoline sales. An estimated two-thirds of the lead exits through the exhaust, and about half of the exhausted lead becomes airborne, that is, each year about 180 million pounds of lead swirls into the atmosphere. Analysis of atmospherically precipitated samples of lead and other metals, collected by a nationwide network of 32 stations throughout the U. S., indicated that the concentration of lead in precipitation correlated with the amount of gasoline consumed in the area in which the samples were collected. The above two investigations suggest that leaded gasolines contribute notably to the environmental (soil and water) burden. Red lead and litharge-based paints were identified as an environmental hazard during shipscraping because of the high temperatures involved in burning off (volatilizing) the spent paint.

Further exploration of the lead-pigment use pattern brought to light the ban on the use of lead pigments in indoor paints. More important, however, it revealed the many instances of pediatric plumbism in substandard housing areas of some of our larger cities, e.g., New York, Baltimore, Cleveland, and Chicago. Factors such as the elimination of lead paint from walls, continuous education and enforcement activities of the city health department, efforts of local pediatric services, establishment of comprehensive health care clinics for children in the susceptible age living in high risk areas, family planning, and an improved social climate, have resulted in a drop of about 50 percent (286 to 141) in pediatric plumbism in Baltimore during two consecutive 3-year periods (1964 to 1966 and 1966 to 1969). Chisolm points out that these corrective measures deserve attention when it is realized that total treatment costs for an institutionalized child suffering from permanent brain damage may amount to approximately $250,000 during a 60-year period.

An examination of the methods of obtaining lead and the diverse uses of lead suggested that the effects of lead on man may be exercised through contact, inhalation, and ingestion. In order to determine the stressor ⟶ source ⟶ transport ⟶ path ⟶ effect (on man) relationship, an environmental stressor matrix was formulated for lead, including lead alloys. The stressor matrix approach appears sufficiently flexible to collect and display information defining the effects that any stressor may exercise, since it permits specification of the medium, the compartment, the portal of entry, target site of action, consequences, and control.

Lead is very slowly absorbed from environmental sources, e.g., soil, water, food, and air, but the gradual accumulation of the element in the body is the basis for progressive toxicity. It was formerly believed that considerable amounts of lead could gain access to the body percutaneously, but this has been refuted; instead, inhalation and ingestion are the two main routes of entry. The average man ingests about 5 mg/yr from drinking water, 100 mg/yr from his diet, and about 15 mg/yr from respiration. Generally, industrial health authorities agree that lead concentration in respired air should be below 150 $\mu g/m^3$ and tetraethyllead (TEL) should be below 50 $\mu g/m^3$. Concentration of lead in ambient air may vary from approximately 0.01 $\mu g/m^3$ to 50 $\mu g/m^3$ (the latter along heavily traveled highways).

Results

The mathematical model developed to represent the environmental transport of lead from source into man is a tool which provides a quantitative investigation of lead concentrations in real world systems, and of particular interest, airborne lead concentrations, foodstuff lead concentrations, and body organ lead concentrations. Since the inhalation and ingestion modes of intake to man are the two major ways in which lead is taken into the body, each of these modes of intake of lead were investigated by utilizing the mathematical model developed for the environmental stressor lead.

The inhalation mode of entry was examined by calculating the concentrations of a airborne lead using Equations (9) through (12) at various distances from four types of sources and at various heights of these sources above the ground surface: (1) a point source, (2) downwind line source, (3) crosswind line source, and (4) area source. A schematic drawing of these sources is shown in Figure 6. A unit emission rate from these sources of 1 g/day, 1 g/m/day, 1 g/m/day, and 1 g/m^2/day, respectively, was used to calculate the airborne lead concentrations due to these sources. Curves for airborne lead concentrations vs distance from the source for each of these sources are shown in Figures 7, 8, 9 and 10 for an average weather condition of turbulence, or mixing, and for a height of 0.25 m above ground surface (the approximate distance of an automobile exhaust above the ground surface). Values for the diffusion coefficients, C, were obtained from Eisenbud(60) while the mean wind speed data were obtained from USPHS Three Cities Study(32). The airborne lead concentrations from any source with similar emission and weather conditions can be calculated from the curves in Figures 7-10 by applying the ratio of actual source emission rate to the unit source emission rate used to generate these curves.

To investigate the airborne lead concentrations in a particular geographical locality, a Midwestern city, Cincinnati, Ohio, was chosen. It was assumed that the major contributor to atmospheric lead in Cincinnati is the exhausts of automobiles. Therefore, the line and area sources were used to calculate airborne lead concentrations due to: (1) automobiles randomly traversing city streets (assuming even distribution over the city) as an area source and (2) automobiles concentrated on a major freeway as a line source (both downwind and crosswind freeways). Curves of airborne lead concentrations vs distance from the source were generated for these three sources for Cincinnati and are shown in Figures 11, 12, and 13.

The emission rates of these sources were calculated as follows:

In the 1969 Market Guide(61) estimates of dollar volume of gasoline sales for 1969 in Cincinnati were $67.9 million. Assuming (a) 3 gallons of gasoline is purchased per dollar of sales, (b) 2.4 grams of lead is contained in each gallon of gasoline, (c) 67 percent of lead consumed in the automobile is exhausted to the atmosphere, and (d) 50 percent of exhausted lead becomes airborne; then approximately one-half million grams of lead per day from automobile exhausts becomes airborne. This quantity distributed over the area of Cincinnati, represents the area source emission rate. Since the wind direction is usually from the south in Cincinnati, the downwind line source emission rate is this quantity of lead released by automobiles each day distributed over the length of the major freeway travelling in a north-south direction while the crosswind line source emission rate is this quantity of lead released by automobiles each day distributed over the length of the major freeway travelling in an east-west direction.